



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

### Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

### About Google Book Search

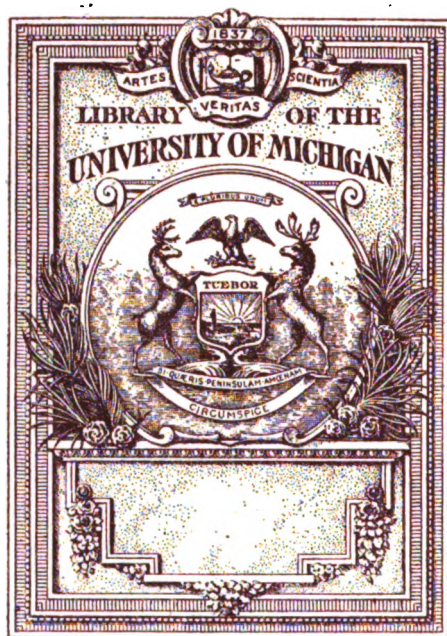
Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>



**B**

3 9015 00208 433 6

University of Michigan - BUHR



610.5  
I 6  
C 64



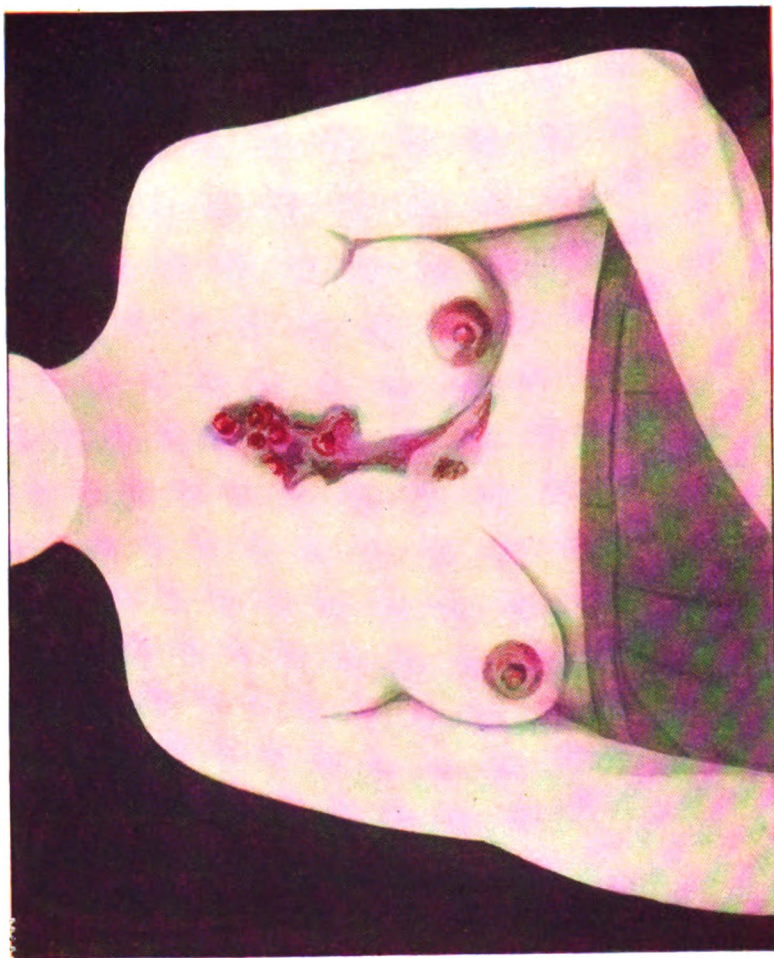








FIG. 1.



Actinomycosis of the breast. Page 30.



# INTERNATIONAL CLINICS

## A QUARTERLY

OF

ILLUSTRATED CLINICAL LECTURES AND  
ESPECIALLY PREPARED ORIGINAL ARTICLES

ON

TREATMENT, MEDICINE, SURGERY, NEUROLOGY, PÆDIAT-  
RICS, OBSTETRICS, GYNÆCOLOGY, ORTHOPÆDICS,  
PATHOLOGY, DERMATOLOGY, OPHTHALMOLOGY,  
OTOLOGY, RHINOLOGY, LARYNGOLOGY,  
HYGIENE, AND OTHER TOPICS OF INTEREST  
TO STUDENTS AND PRACTITIONERS

BY LEADING MEMBERS OF THE MEDICAL PROFESSION  
THROUGHOUT THE WORLD:

EDITED BY

H. R. M. LANDIS, M.D., PHILADELPHIA, U.S.A.

WITH THE COLLABORATION OF

CHAS. H. MAYO, M.D.

ROCHESTER

SIR JOHN ROSE BRADFORD, M.D.

LONDON

HUGH S. CUMMING, M.D., D.P.H.

WASHINGTON, D. C.

WILLIAM S. THAYER, M.D.

BALTIMORE

JOHN G. CLARK, M.D.

PHILADELPHIA

FRANK BILLINGS, M.D.

CHICAGO

JAMES J. WALSH, M.D.

NEW YORK

A. MCPHEDRAN, M.D.

TORONTO

CHARLES GREENE CUMSTON, M.D.

GENEVA

J. W. BALLANTYNE, M.D.

EDINBURGH

JOHN FOOTE, M.D.

WASHINGTON, D. C.

WITH CORRESPONDENTS IN MONTREAL, LONDON, PARIS  
AND GENEVA

---

VOLUME III. THIRTY-FIRST SERIES, 1921

---

PHILADELPHIA AND LONDON

J. B. LIPPINCOTT COMPANY

1921

**COPYRIGHT, 1921**  
**BY**  
**J. B. LIPPINCOTT COMPANY**

**PRINTED BY J. B. LIPPINCOTT COMPANY, PHILADELPHIA, U. S. A.**

## CONTRIBUTORS TO VOLUME III

### (THIRTY-FIRST SERIES)

---

- ARIBAUD, GEORGE, M.D., Lyons, France.
- BEHAN, R. J., M.D., F.A.C.S., Surgeon to the St. Joseph's Hospital, Pittsburgh, Pa.
- BÉRARD, D. G., M.D., Navy Medical Service, Bordeaux, France.
- BETTMAN, RALPH B., M.D., Adjunct Surgeon to the Michael Reese Hospital, Chicago.
- BONNET, DENIS, M.D., Former Interne of the Hospitals of Lyons, France.
- COULTER, JOHN S., M.D., F.A.C.S., Formerly Lt. Colonel, Medical Corps, U. S. Army, Consulting Surgeon, Nokomis Coal Co.
- FREEMAN, ROWLAND G., M.D., New York City.
- FOSTER, G. S., M.D., Surgeon to Notre Dame Hospital, Manchester, N. H.
- HAMMERSCHLAG, VICTOR, M.D., Vienna.
- HINGLAIS, MANUEL, M.D., Formerly Resident Physician to the Hospitals of Lyons and of Bône, France.
- JOB, EMILE, M.D., Military Medical School, Lyons, France.
- JOPSON, JOHN H., M.D., Surgeon to The Presbyterian and Children's Hospitals; Professor of Surgery, Graduate School, University of Pennsylvania, Philadelphia.
- KEILTY, ROBT. A., M.D., Department of Laboratories and Research Medicine, The Geisinger Memorial Hospital, Danville, Pa.
- MAGNUSON, PAUL B., M.D., Medical Director, Illinois Industrial Commission; Attending Surgeon, Wesley Hospital and Alexian Brothers' Hospital; Instructor in Surgery, Northwestern University Medical School, Chicago.
- MILIFF, S., M.D., Breznik, France.
- MONESTIÉ, FRANÇOIS, Formerly Interne of the Hospitals of Lille; Member of the Anatomical Clinical Society of Lille, etc., France.
- NAUSSAC, JOSEPH, M.D., Faculty of Medicine of Lyons, France.
- PFEIFFER, DAMON B., M.D., Associate in Surgery, University of Pennsylvania, Philadelphia.
- POITEAU, M., M.D., From the Pathological Department of the University of Lille, France.
- RAVDIN, ELIZABETH G., Philadelphia.
- RAVDIN, I. S., Philadelphia.
- REYNOLDS, CECIL EDWARD, Los Angeles, Calif.
- ROBERT, GEORGE P., Formerly Assistant at the Ophthalmological Clinic of the Faculty of Medicine, Lille, France.
- WALSH, JAMES J., M.D., Ph.D., Medical Director of Fordham University, School of Sociology; Professor of Physiological Psychology at Cathedral College, New York City.
- WEBER, F. PARKES, M.A., M.D., F.R.C.P., London, Eng.



# CONTENTS OF VOLUME III

(THIRTY-FIRST SERIES)

## SYMPOSIUM ON ACTINOMYCOSIS

PAGE

THE PATHOLOGY, SYMPTOMATOLOGY AND DIFFERENTIAL DIAGNOSIS OF PULMONARY ACTINOMYCOSIS. By JOSEPH NAUSSAC, M.D. ....	1
THE PATHOLOGY OF ACTINOMYCOSIS OF THE BREAST. By M. PORTEAU, M.D. ....	19
THE CLINICAL ASPECTS OF ACTINOMYCOSIS OF THE BREAST. By S. MILEFF, M.D. ....	29
ACTINOMYCOSIS OF THE TONGUE, WITH ILLUSTRATIVE CASES. By DENIS BONNET, M.D. ....	34
THE HEPATIC LOCALIZATIONS OF ACTINOMYCOSIS. By GEORGE ARIBAUD, M.D. ....	50
ACTINOMYCOSIS OF THE CÆCUM AND APPENDIX. By MANUEL HINGLAIS, M.D. ....	59
ACTINOMYCOSIS OF THE SKIN. By FRANÇOIS MONESTIÉ, M.D. ....	73
ACTINOMYCOSIS OF THE LACHRYMAL DUCTS. By GEORGE P. ROBERT, M.D. ....	83
ACTINOMYCOSIS OF THE NERVOUS SYSTEM. By EMILE JOB, M.D. ..	90
THE TREATMENT OF ACTINOMYCOSIS WITH POTASSIUM IODIDE. By D. G. BÉCARD. ....	107

~~THE TREATMENT OF ACTINOMYCOSIS WITH POTASSIUM IODIDE. By D. G. BÉCARD. ....~~

## CLINICS

ON ABSCESS OF THE PREVESICAL SPACE AND UMBILICUS, WITH SPECIAL REFERENCE TO THEIR ORIGIN FROM CYSTS OF THE URACHUS, AND REPORT OF A CASE SIMULATING URACHAL CYST. By DAMON B. PFEIFFER, M.D. ....	111
MECKEL'S DIVERTICULUM INCARCERATED IN AN INGUINAL HERNIA. By RALPH B. BETTMAN ....	126

## MEDICINE

THROMBOSIS OF THE INFERIOR VENA CAVA AND BOTH RENAL VEINS. By F. PARKES WEBER, M.A., M.D., F.R.C.P. ....	132
HEART BLOCK SIMULATING PERFORATED PEPTIC ULCER. By I. S. RAVDIN, B.S., M.D. AND ELIZABETH GLENN RAVDIN, A.B., M.D. ....	138
NEUROSES AND PSYCHONEUROSES AND THE THERAPEUTIC VALUE OF FOOD. By JAMES J. WALSH, M.D. ....	156

v



**PÆDIATRICS**

<b>DISTURBANCES OF HEARING IN CHILDREN AND THEIR INFLUENCE ON THEIR DEVELOPMENT. BY PROFESSOR DOCTOR VIOTOR HAMMERSCHLAG .....</b>	<b>175</b>
<b>PÆDIATRICS CLINIC. BY ROWLAND G. FREEMAN, M.D. ....</b>	<b>189</b>

**INDUSTRIAL MEDICINE**

<b>EDITED BY PAUL B. MAGNUSON, M.D. AND JOHN S. COULTER, M.D., F.A.C.S. ..</b>	<b>195</b>
--	------------

**SURGERY**

<b>THE REDUCTION OF FRACTURES OF THE LOWER END OF THE RADIUS. BY JOHN H. JOPSON, M.D. ....</b>	<b>250</b>
<b>THE RADICAL CURE OF HYDROCEPHALUS. BY CECIL EDWARD REYNOLDS, M.R.C.S. ....</b>	<b>255</b>
<b>FRACTURES OF THE SKULL—MECHANISM OF THEIR PRODUCTION. BY R. J. BEHAN, M.D. ....</b>	<b>259</b>
<b>PHYSIOLOGICO-PATHOLOGICAL DISCUSSION IN A SYMPOSIUM ON THE ACUTE ABDOMEN. BY ROBT. A. KELTY, M.D. ....</b>	<b>273</b>
<b>THE SURGICAL KIDNEY. BY G. S. FOSTER, M.D. AND S. MILLER, M.D. ..</b>	<b>281</b>

# LIST OF ILLUSTRATIONS TO VOLUME III

## (THIRTY-FIRST SERIES)

### COLORED PLATES

PAGE

Actinomycosis of the breast .....	<i>Frontispiece</i>
After curetting away the granulation tissue, all the incisions were united by drains .....	68

### PLATES

Figure 1 .....	2
Figure 2 .....	2
Figure 3 .....	2
Section of breast enlarged twice its actual size (Fig. 1) .....	22
Young nodule (x860) (Fig. 2) .....	22
Old nodule (x860) (Fig. 3) .....	23
Section of the mammary gland including the nipple (x50) (Fig. 4) .....	23
Lateral view showing scaphoid epigastrium, protruding umbilicus and hypogastric prominence of the subcutaneous abscess (Fig. 1) .....	112
Cystoscopic appearance of hillock seen at the summit of the bladder at the vesical terminal of the urachus (Fig. 2) .....	112
Schematic representation of the site of the abscess (Fig. 3) .....	113
Sagittal frozen section of a female cadaver after artificial infiltration of the tissues with water by arterial injection under pressure of about 10 feet (Fig. 4) .....	116
Urachus with cystic dilatations at vesical end (Fig. 5) .....	120
Urachus cyst (Fig. 6) .....	121
Delore and Cotte's case of intraperitoneal cyst of the urachus (Fig. 7) ....	124
Meckel's diverticulum (Fig. 1) .....	129
Thrombosis of the inferior vena cava (Fig. 1) .....	134
Scheme of the general arrangement of the Autonomic Nervous System (Fig. 1) .....	139
Cutaneous sensory distribution of spinal nerves (Fig. 2) .....	147
Physiotherapy in Nokomis, Illinois, coal mining town (Fig. 1) .....	202
Physiotherapy in the coal mining industry (Fig. 2) .....	202
Physiotherapy Laboratory at the Illinois Steel Corporation (Fig. 3) ....	203
Physiotherapy in the Steel Industry (Fig. 4) .....	203
Physiotherapy in the Illinois Steel Corporation (Fig. 5) .....	204
Pneumoconiosis (Fig. 6) .....	210
Sarcoma of the femur (Fig. 7) .....	220
Fracture of the acetabulum with internal dislocation of the femur (Fig. 8) ..	224

"Skid" for reduction of dislocated semilunar bone (Fig. 9) .....	226
Lateral view of "skid" (Fig. 10) .....	226
Diagram showing the normal relationship of the semilunar bone to the radius and the os magnum (Fig. 11) .....	226
Diagram showing the relationship of the dislocated semilunar bone to the radius and the os magnum (Fig. 12) .....	226
Diagram showing the "skid" and its relationship to the dislocated semilunar bone (Fig. 13) .....	227
Showing a dislocated semilunar bone (Fig. 14) .....	226
Showing the same wrist as in Fig. 6 after reduction by open operation (Fig. 15) .....	227
First step in reduction (Fig. 1) .....	251
Second step in reduction (Fig. 2) .....	252
Third step in reduction (Fig. 3) .....	253
Typical Colle's fracture before reduction (Fig. 4) .....	254
Same fracture after reduction (Fig. 5) .....	254
The patient shortly after diphteria (Fig. 1) .....	256
As he was July 21, 1920 (Fig. 2) .....	256
Close-up of face shows paralysis of tongue and jaw (Fig. 3) .....	256
Radiograms showing the openings through which the intra-muscular tissue drains were fashioned (Fig. 4) .....	257
Somnolent paralysis shortly after final operation (Fig. 5) .....	257
The first successful effort at concerted movement of all four limbs on the fifth post-operation day (Fig. 6) .....	257
The fatuous semi-paralytic stage (Fig. 7) .....	256
Early acts of intelligence about the tenth day (Fig. 8) .....	256
Also note the defective visions before the discs cleared up (Fig. 9) .....	256
Early efforts in speech (Fig. 10) .....	257
August 28th, one month after operation (Fig. 11) .....	257
The incisions from left side (Fig. 12) .....	257
The cross-bow incision (Fig. 13) .....	257
The patient on November 10, 1920 (Fig. 14) .....	256
The arachnoid covering the fourth ventricle and its pial diaphragm (Fig. 15) .....	257
Case photographed in the Clara Barton Hospital (Fig. 16) .....	256
Same girl as Fig. 16 a few weeks after the deduralization (Fig. 17) .....	257
Same girl as Fig. 16. This girl has developed generalized tuberculosis and is near death (Fig. 18) .....	256
Same girl as Fig. 16 a few weeks after the deduralization (Fig. 17) .....	257
The other patient presented two years after operation (Fig. 20) .....	257
Photo taken October, 1920, of E. P. (Fig. 21) .....	256
The attitude in paralysis of the anterior vermis (Fig. 22) .....	257
Distension of the third ventricle (Fig. 23) .....	256

# LIST OF ILLUSTRATIONS TO VOLUME III

ix

Gutter fracture (Fig. 1) .....	260
Bursting fracture (Fig. 2) .....	260
Illustration taken from Manual of Neuro-Surgery, showing the meridional lines of fracture (Fig. 3) .....	260
Three rotating movements of a bullet in flight (Fig. 4) .....	261
Normal skull (Fig. 5) .....	262
Shows the result of a bullet perforating the skull (Fig. 6) .....	263
Bullet wound of the skull (Fig. 7) .....	260
Shows a fracture and depression of the internal table of the skull (Fig. 8) ..	260
Illustrates what happens in the skull when a small object, such as a bullet, comes in contact with the skull .....	264
Shows a depressed skull (Fig. 10) .....	265
So called ping-pong fracture of the skull (Fig. 11) .....	266
Appearance of external surface of skull (Fig. 12) .....	266
Inner surface of skull (Fig. 13) .....	266
Radiograph of skull (Fig. 14) .....	266
View of skull by transmitted light .....	268





# Symposium on Actinomycosis

---

## THE PATHOLOGY, SYMPTOMATOLOGY AND DIFFERENTIAL DIAGNOSIS OF PULMONARY ACTINOMYCOSIS

By JOSEPH NAUSSAC, M.D.

Faculty of Medicine of Lyons, France

---

*Pathology.*—From the clinical viewpoint we shall see that pulmonary actinomycosis presents rather disparate symptoms, but as Pic has pointed out: "The various forms correspond more to the clinical evolutions than to pathological differences." This is due to the way in which the actinomyces act in the lung. It almost invariably invades the connective tissue in preference to others, and then extends like a mole in his subterranean tunnels. A considerable hyperplasia of the interstitial tissue ensues which surrounds and chokes the parenchyma, a sclerosis, a cirrhosis of the organ ensues, and is the widest of the parenchyma whose cells degenerate from the action of the pyogenic power of the parasite or with the help of the associated bacteria, inflammatory areas develop whose evolution almost always ends in caseation or purulent disaggregation. It is for this reason that almost all the different parts of the lung are in turn involved, no matter what may be the starting-point of the process. Bronchial tubes, vessels, pulmonary parenchyma, interstitial connective tissue and pleura are successively involved and it is to all these different lesions that we must now turn our attention. For the better understanding of the subject I shall describe three forms of the disease, namely, *broncho-actinomycosis*, *pneumo-actinomycosis*, and *pleuro-pneumo-actinomycosis*. Choux adds a fourth form, *thoraco-pulmonary actinomycosis*, but I would point out that this process does not confine its action to the lungs and pleura, but invades the thoracic walls by continuity, in the same way as the diaphragm, pericardium, and vertebrae. Therefore this fourth type enters into the study, not of pulmonary actinomycosis, but in that of thoracic actinomycosis and should from our viewpoint be regarded as a complication of the pulmonary process.

But since the pleura almost always participates to some extent in the pulmonary process, I shall take for my description the pleuro-pulmonary form. The lesions vary from one case to another, and one observes successively in the same subject broncho-pneumonia, epithelial pneumonia, interstitial pneumonia and granulia, which, however, is different from acute tuberculosis, while the pleuresy varies from a simple serofibrinous collection to pleural empyema.

At autopsy, the involved lung is not merely adherent but, so to speak, glued to the thoracic wall, clavicle and pericardium. These adhesions are so intimate that they cannot be broken down without tearing the pulmonary tissues and are the products of connective tissue hyperplasia.

The pleurae are thickened, while the visceral surface gives off bands of fibrous tissue extending to and entering the lung, where they give rise to trabeculae which, by dividing, surround the lobules, alveolae, and cells, finally choking the latter. This lesion merely represents one very frequent form of the process, but it is not constant. Thus, in Fig. 1, the section of the lung shows epithelial pneumonia but no interstitial lesions.

The pleurae are thickened and there may be fluid collection, often considerable in amount, which can be detected by auscultation during life. In one case the fluid was serofibrinous, probably due to an irritative process in the neighborhood. In most cases the collection is purulent; in one, it communicated by a pleuro-pulmonary sinus with an intra-pulmonary abscess.

It is a question whether or not the pleuresy is purulent from the onset. Although this possibility cannot be denied it is perhaps probable that the irritation due to pulmonary inflammation produces a fibrinous collection which later becomes purulent, as occurred in a case reported by Henck. But in many instances the pleurae collection should be regarded less as a pleuresy than as the contents of a pulmonary or thoracic abscess that has ruptured into the pleurae cavity.

The lungs are thickened, oedematous and hard, offering the picture of disseminated hepatization. Sometimes they are increased in size and seem to be too large for the thorax. In other cases they are atelectatic, brown and violet color, with a smooth surface. The vessels are gorged with blood.

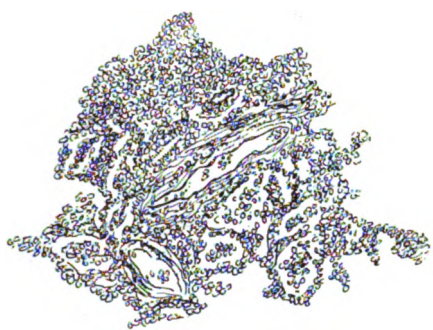


FIG. 1.



FIG. 2.

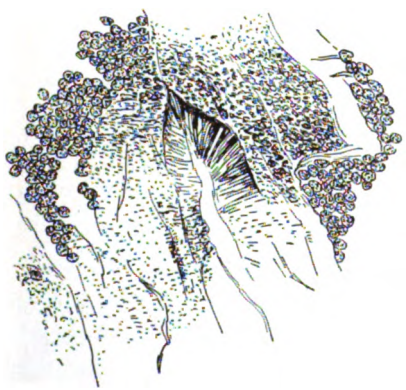


FIG. 3.



When, on the contrary, lesions of sclerosis predominate, the lung is small, retracted toward the hilum. Pulmonary insufficiency then ensues, just as there is hepatic insufficiency in cirrhosis or renal insufficiency in Bright's disease. Let it be added that all these lesions may be present in the same lung, as will be seen by Fig 2. Besides the areas of intense inflammation, neoplastic tissue can be detected in the microscopic field, resembling sarcoma.

In the midst of this altered pulmonary tissue the areas of broncho-pneumonia will be found, as well as more or less large abscesses; some in the phase of simple inflammation, others undergoing purulent disintegration or caseation. In these miliary abscesses or in the cavernulæ or cavities to which they give rise, grains will be found which microscopically are the actinomyces. The mycelium is not very apparent but conidia will be perceived on the contour of the specimen. I here give a drawing (see Fig. 3) of one of these miliary abscesses with a typical grain in the centre.

In this drawing, in the midst of a very pronounced zone of hepatisation which becomes attenuated, one will see, if the specimen is examined from the centre to the periphery, tracts of connective tissue passing through the pulmonary cells. In the centre of the preparation is a grain of actinomycosis whose microscopical aspect has been damaged by the manipulations in preparation. The preparation stained by picro-carmin does not show the mycelian filaments but the conidia are distinctly seen. It is a miliary abscess in the midst of the pulmonary parenchyma.

Fig. 2 shows the usual lesions of pulmonary actinomycosis. It was procured from one of our cases in Professor Poncet's service. The visceral pleura is very thick, and in a corner of the drawing fibrous tissue tracts are seen arranged in rays going to the centre of the lobule, surrounding the pulmonary lobules, alveolæ and even the cells of the lung. The aspect of this neoplastic zone is not uniform. In spots embryonal fusiform or stellate cells seem to be arranged in a certain direction, recalling a spindle-cell sarcoma. These embryonal cells possess large nuclei, and from their poles rather long and large prolongations are given off which anastomose with those of other cells or become lost in the hyperplastic connective tissue. In the midst of the latter the cells of the pulmonary paren-

chyma have disappeared, and in only one or two spots can pigment deposits be detected.

Along side this zone and without any appreciable line of demarcation, the picture is somewhat different. Instead of being elongated, the cells become large, rounded and present a large nucleus having a striated aspect. A very fine amorphous substance exists between them. The vessels are somewhat more numerous here.

In other words, the first preparation assumes the aspect of a spindle-cell sarcoma, while in the second preparation described, it resembles a lymphadenoma.

If another part of the preparation be examined the hyperplastic lesions will be found to have become inflammatory. The pulmonary tissue offers hepatization. Nevertheless, the connective tissue is very abundant, but zones of inflammation are seen in the centre of the lobule. The cells of the bronchial walls are cloudy, their nuclei cannot be detected, while in spots there is desquamation of the epithelium. The lumen of the bronchial tubes is filled with leucocytes, and around the tubes the pulmonary tissue has undergone hepatization. In the centre of these spots it has even commenced to become purulent, while at the periphery the inflammation finally becomes attenuated. Otherwise put, the process is one of true miliary abscess formation in the midst of the lung. It was in one of these abscesses that I obtained the actinomycotic grain already described.

In Germany an attempt has been made to make a differential pathologic diagnosis of the affections with other infectious diseases. Ziegler studied the broncho-pneumonia of actinomycosis, syphilis and glanders. He states that actinomycosis produces pulmonary lesions which much resemble a large focus of broncho-pneumonia. In oxen hard foci are found of a granular type, their centre being occupied by a fungus. In man the affection assumes very special character. Small foci of yellow grains, or grayish-yellow grains, are found. They maintain a little suppuration in the tissues and give rise to some cavities. This suppuration continuing for sometime, larger cavities develop and finally the pleura becomes involved. In order to recognize actinomycosis it is only necessary to search for the actinomyces in the pus or pathologic or infiltrated pulmonary tissues.

The bacilli of glanders when located in the lung present small

granular foci usually located directly under the pleura. At first they are rather soft and grayish-white in color, then they become hard and almost white. Beside these lesions large granular foci are seen, pinkish or grayish in color.

In pulmonary syphilis a bronchitis finally develops during which symptoms of bronchial stenosis or broncho-pneumonia appear.

In Fig. 1 it will be noted that even in the midst of an abscess undergoing purulent melting the vessels are absolutely intact, and I might say that this is *almost* characteristic of actinomycosis. At spots they are gorged with blood, at others they contain a few red blood corpuscles, according to the state of congestion of the parts observed. This is a differential sign, because in pulmonary tuberculosis the vessels are not respected by the morbid process and this explains why hemoptysis is infrequent in actinomycosis of the lungs.

The preparation just referred to represents the typical form of lesions of pulmonary actinomycosis. Very marked interstitial lesions and formation of embryonal tissue; in a word association of neoplastic tissue, or at least hyperplastic, and areas of the inflammation. In these circumstances the actinomyces alone produced the lesions, no bacteria had become associated. The tubercle bacillus is absent and the fungus in order to penetrate into the lung followed the connective tissue, hence the special form of the lesions.

It was quite different in the case I will report later, (case 1). The invasion of the lung by the actinomyces followed a lesion of the larynx, the lung being invaded by way of the bronchial tubes. The lesions (see Fig. 1) are more epithelial than interstitial, the pulmonary parenchyma being particularly involved. There is no tissue resembling neoplastic tissue; the process is distinctly inflammatory.

The bronchial tubes having been the point in the lungs primarily attacked the evolution of the process began by bronchitis. This in turn extended, reached the bronchioli and then the capillary bronchitis transformed into a broncho-pneumonia. This was the predominating lesion. The bronchial tubes presented a considerable proliferation of their epithelial cells which became vesicular and underwent segmentation. The connective tissue was infiltrated with leucocytes and predominated in the structure of the walls of the bronchial tubes. Their lumen was occluded by all the products of disquamation—leucocytes, fibrinous exudates, etc.

The blood-vessels were gorged with blood and were not free as in the case referred to above which denotes the greater intensity of the inflammatory phenomena. Areas of hepatization and splenization were found side by side, with peribronchial nodules in all the phases of their evolution. At spots these nodules had undergone or were commencing purulent melting, the cells degenerated, caseation developed, but no giant cells could be discovered.

Briefly stated, this case presented lesions produced by ordinary broncho-pneumonia and were it not for the integrity of the walls of the bronchial tubes, presented nothing special or characteristic of actinomycosis.

The reason for the difference in the type of lesions in different cases resides in the association of bacteria. The pathognomonic lesions of actinomycosis of the lungs are: Hyperplasia of the interstitial connective tissue causing the aspect of a neoplasm-sarcoma, lymphadenoma associated with acute inflammatory phenomena, the integrity of the vessels and absence of giant cells. Now that the lesions or pulmonary actinomycosis have been described we can consider the signs by which they manifest themselves. I will begin by relating the clinical history of the case referred to above as it has many points of interest.

*Case 1.*—Male, *æt.* forty-five years, married, has had four children, only one living but in good health, the three others died between the ages of five months and one year. His wife had a miscarriage from which she died.

Personally he has never been ill. Denies syphilis, rheumatic fever or pulmonary affections. Lost one eye from an accident. Ten years ago an abscess of the right molar tooth was incised; cicatrix still visible and adherent to bone.

Present illness dates back thirteen months. At that time he felt some stinging sensation in the supra-hyoid region and a few days later a painless tumefaction developed near the right wing of the thyroid cartilage. This progressively increased until it reached the size of a pigeon's egg. No dysphagia, no dyspnoea. General health unaffected.

At this time a laryngological examination revealed considerable œdema of the right aryteno-epiglottic fold, and this extended over the entire arytenoid cartilage and inferior vocal cord. The entire



larynx was dark red. No ulcer. The diagnosis wavered between syphilitic or infectious laryngitis but there was no question of a neoplasm.

On October 21, the patient returned to the hospital, after an iodide treatment without any benefit. The tumefaction was now fluctuating, and the collection was pointing under the skin. An incision gave exit to a serous fluid in which search for the actinomyces was neglected.

Three months later the tumefaction had recurred at the same spot. A longer incision was this time made but in a few weeks the lesions began to extend along the right sternomastoid muscle which was surrounded by an indurated pastey mass, the middle cervical space on the left then became infiltrated so that the case began to look like a laryngeal neoplasm which was invading the surrounding parts. However, a laryngeal examination was negative and again the collection was incised and the iodine treatment continued.

The patient's general condition greatly improved so that he was sent to a convalescent home in August, but on November 2, he was admitted again to the hospital as an urgent case. For several days before admission he had been seized by severe attacks of suffocation which might require tracheotomy.

On the patient's entrance he was cyanosed and appeared to be on the point of asphyxia. For two days everything was kept in readiness for tracheotomy, but by rest this acute phase finally subsided so that at the end of a week the patient was in a condition for a more thorough examination. The head was as if glued to the thorax by a plaster of wooden hardness extending from one sternomastoid muscle to the other; the anterior cervical region was lumpy and presented several sinuses adherent to the deep structures, some being in the infrahyoid region, the others lying along the sternomastoid muscles.

All the tissues were hard and the various structures of the neck were matted together, but by transition those of the neck were about normal. However, below the right occipital line a tumor the size of a small egg was noted, superficially immovable and presenting a sinus with depressed and adherent edges. All the sinuses were of little depth and had no apparent connection with the skeleton; they

gave exit to a granular magma. Spontaneous movements of the head were very limited in every direction, especially flexion.

By auscultation crepitant râles were heard behind over the left apex; respiration was rough while in both apices; in front and behind the vesicular murmur was not heard on account of laryngeal tugging. Sibilant râles were heard over the front of the right base.

The voice was almost extinguished, the dyspnoea considerable. Laryngological examination did not reveal any deviation or deformity of the larynx, but there was a general infiltration of the aryteno-epiglottic folds and the upper cords, which were swollen, and covered by a layer of muco-pus. No paralysis of the cords. The digestive functions were normal although deglutition of food required much time. Exploration of the œsophagus with an olive pointed sound No. 2 was stopped at the entrance of the gullet by an obstruction and all attempts to pass by it produced intense laryngeal reflex. Professor Jaboulay who now saw the patient recalled the history of the case and at once eliminated malignant disease and made the diagnosis of actinomycosis.

For some weeks the patient was given from four to six grammes KI daily, and the signs of chronic phlegmon diminished but dysphagia appeared and feeding became difficult. Emaciation was marked and death seemed near to hand.

*Autopsy.*— On opening the thorax a serofibrinous fluid without yellow grains or any elements of actinomycosis was found in the right pleura. The corresponding lung, greatly retracted toward the hilum, was completely infiltrated by chronic inflammatory lesions with two cavities with fibrous walls in the region of the upper border of of the upper and middle lobes. No other lesions were found.

The left lung was œdematous with a few foci of bronchopneumonia and quite a free flow of pus from the bronchial tubes.

The pericardium was dry without any characteristic change of the serosa. The thoracic and abdominal portions of the digestive track were normal. The spleen was somewhat enlarged and sclerous. Both kidneys presented chronic inflammatory changes. They were pale, slightly enlarged and the vascular striations of the cortex altered. But nothing was seen recalling the lesions of actinomycosis.

The larynx, pharynx and the upper part of the œsophagus with two vascula-nervous bundles of the neck were embedded in a mass

of lardaceous inflammatory tissue throughout which yellow puriform masses were scattered but without any grains of actinomycosis. This inflammatory tissue solidly fixed the vessels to the adjacent organs, especially on the right, but did not compress them to the extent of occlusion. During life there had been no symptoms of interference the cerebral circulation. Both lobes of the thyroid, infiltrated with the same tissue, were glued to the larynx, and it was the pressure of this combined mass that produced the pharyngo-oesophageal stenosis, causing obstruction to the passage of the sound and the dysphagia. There was no organic stricture of the gullet whose mucosa was only perforated by a sinus connecting it with the foci in the middle cervical space.

The mucosa of the larynx was intact but infiltrated in all the parts lined by loose connective tissue—epiglottic folds, upper vocal cords and ventricles. This chronic oedema explained the congestive accident occurring in sudden paroxysms. No deformity of the cartilages.

Histological examination confirmed the data obtained by microscopic examination. In no tissue could the mycelium or conidia of actinomycosis be discovered. Only chronic inflammatory lesions with fibrous tissue neoformation could be found with areas of leucocytic infiltration in the perivascular regions, forming true miliary abscesses. The two pulmonary cavities offered the same aspect; neither giant cells nor Koch's bacillus could be detected.

It is evident that if one is in presence of a patient with actinomycosis of the larynx, oesophagus or face, for example, and the process extends to the lungs, the pulmonary symptoms will be overshadowed or at least complicated by symptoms belonging to actinomycosis in general. But besides the general phenomena and the signs found by auscultation upon which I shall speak, there will be the three symptoms present, namely, early trismus, very sharp pain and the development of a tumor. These symptoms need not be considered since they evidently have nothing to do with primary actinomycosis of the lung, which forms the subject of this paper.

It is rather difficult to estimate the duration of the incubation period, even approximately and opinions differ considerably. Dor and Bérard, inoculated the actinomyces in the vitreous body of a rabbit's eyes and found that yellow grains had already formed in the

anterior chamber at the end of a week. At autopsy of the animal very numerous pleural adhesions were found and a cavity at the pulmonary apex of one lung containing a finely aerated creamy pus. But it was difficult to say just when the pulmonary lesions first developed.

From the clinical viewpoint I shall divide the process into three forms, namely, *broncho-actinomycosis*, *pneumo-actinomycosis* and the *pleuro-pulmonary form*.

*Broncho-actinomycosis*.—This form is the rarest of all, therefore the most difficult to diagnose. It is perhaps this reason why so few cases have been reported, since only the characters and microscopic examination of the sputum will reveal the true nature of the process. In a case reported by Canali, the patient was a girl of fifteen whose illness seemed to be a catarrhal bronchitis. But it was not long before suspicion arose as to the nature of the affection on account of the nature of the sputum which was horribly fetid, viscous, yellow and contained little greenish masses. By rest, the sputum separated into two layers; the upper rich in clear mucus, the lower viscous, composed by a yellow sediment in which numerous elements of the actinomyces were found. Antiseptic treatment diminished, although it did not abolish, the fetidity of the expectoration. The colonies of actinomyces became fewer and fewer so that considerable improvement ensued, but the ultimate outcome is not known as the patient was lost sight of.

Neither auscultation nor percussion gave any indication as to the state of the lungs, only sibilant and subcrepitant râles could be detected. This case is at least curious, and it is unfortunate that details are wanting. In the first place, the duration of the disease—eight years—is certainly peculiar and it is hard to believe that the fungus, sufficiently vivacious to live so long, should not have attacked the lung, since it is known that the lesions extend by continuity. However, nothing shows that the pulmonary structures were not involved, the lesions being so small that they did not give rise to symptoms and Poncet has pointed out that subacute actinomycosis assumes a torpid chronic course.

*Pneumo-actinomycosis*.—This form frequently has a violent onset, recalling that of acute pneumonia, with rusty-colored, tenacious sputum. But usually the onset is slower and more insidious and is

preceded by general phenomena. Often a dry cough slowly develops, especially noticeable in the morning. The patient will have noticed that he has been losing flesh for some time, as well as strength and appetite. In Canali's second case the patient was seized with accidents in the midst of perfect health, so that the attending physician thought the cough was due to a commencing pregnancy.

Pyrexia will be noted, especially in the evening, and nocturnal sweats as well. This rapid onset recalls very closely that of pulmonary tuberculosis and the differential diagnosis will be very difficult indeed if examination of the sputum does not give any data. This phase of the process is interrupted by acute phenomena to which the commencement of the illness is usually attributed by the patient. It may be a bronchitis, broncho-pneumonia, pleuresy, or fever and bronchitis. These various affections develop suddenly, progress normally, and then instead of subsiding the patient continues to lose flesh, coughs and becomes cachectic so that a diagnosis of tuberculosis is made until other symptoms arise indicating the true nature of the case. In a case observed by Lumniezer, hemoptyses had occurred for several years, which naturally complicated the diagnosis. But this symptom is rather rare, because as I pointed out when speaking of the pathology, the vessel walls are always intact even when surrounded by a focus of pus.

Following this rather usual onset, constituted especially by malaise of different kinds the symptoms properly belonging to pulmonary actinomycosis appear. The patient, who was supposed to have had frank pneumonia or ordinary serofibrinous pleuresy, improves. The symptoms subside and from an acute process the affection becomes chronic. Dyspnoea is frequently noted but it is, however, rarely intense. It is rather a continued oppression, than otherwise, the patient has difficulty in breathing. At this time certain symptoms may lead one to suspect the nature of the process.

I would first refer to the paroxysmal pain, true fulgurant pain, complained of in the thorax. The radiations may be very severe and often occur at the onset. Then these phenomena give place to a duller and continued pain, a sensation of constrictions. The side involved may be painful to pressure. In one of Netter's patients fleeting pains were felt in the left side of the thorax although the patient appeared to be otherwise in perfect health. Then the pain became fixed, respi-

ration became frequent and difficult, cough developed and the process became established.

The pain is often caused by mediastinitis often wet within pulmonary actinomycosis.

The facies offers nothing special, resembling if anything that of a tuberculous patient. Emaciated, without strength, the color pale, the cheeks hollow and the cheek bone flushed, the patient remains seated, the body bent forward, the arms leaning on an object in order to ease the action of the inspiratory muscles. The voice is low, the respiratory movements traversed by more rapid and prolonged respirations.

Then a dry, painful cough appears, especially in the morning and evening, but later becomes moist. The sputum at first viscous, becomes filled with small greenish purulent masses, which adhere to the bottom of the jar, odorless and offering fine striations of blood. If the sputum be examined one will perceive that it contains little grayish grains of considerable consistency since they can be dissociated from the tissues and organic detritus surrounding them. When examined microscopically the presence of the characteristic mycelium will be seen.

Fever is usually moderate, less sharp than in pulmonary tuberculosis, with vesperal increase and morning remission, but the oscillations of the thermic curve are less considerable than in phthisis. Pyrexia often occurs at the onset of the disease at a time when the lesions have hardly begun. It is probably due to successive outbursts resulting from the development of the parasite and above all to bacterial associations as well as to their toxins.

What is most striking about the fever is that in most published cases typhoid fever has been noted in the patient's antecedents. In Reboul's case the patient developed typhoid a year after the first symptoms and one month after, convalescence became established. Macaigue's patient gave the history of a "mucous fever" two years before the eclosion of the pulmonary actinomycosis, and other similar instances have been recorded. It is therefore to be questioned whether or not there was a true typhoid or an intestinal form of actinomycosis, similar to the intestinal disturbances with pyrexia met with at the onset of tuberculosis. And although this seductive theory

may not be true, the coincidence of the two processes is none the less curious.

Such disparate and varied lesions as are produced by actinomycosis of the lungs naturally do not give rise to distinctive symptoms on auscultation. It can readily be conceived that lesions due to broncho-pneumonia will be accompanied by signs belonging to this process and the same applies to lesions of pneumonia. So that there are no pathognomonic signs of pulmonary actinomycosis, and at the most they will be slightly changed by the lesions belonging to this disease. The actinomyces progressively extending and preferably following the interstitial connective tissue will give moderate and indefinite stethoscopic signs and at autopsy one is astonished to find very advanced lesions with symptoms so little marked.

Palpation shows a decrease of the thoracic vibrations, while percussion reveals subdullness over the entire area involved. It is usually the middle and lower lobes that are involved as evident difference with what occurs in the tuberculosis in which the apex is most frequently the first involved.

On auscultation there is absence of vesicular murmur or at least it will be diminished. Respiration is rough, prolonged with sibilant and subcrepitant râles, intermingled with bronchial souffle. Bronchophony is sometimes present. The respiratory roughness may be replaced by souffles without either râles or friction sound.

The pulmonary lesions often assume the form of lobar pneumonia, therefore the symptoms will be similar to those belonging to this affection. At the onset subdullness is more complete, the side is painful and the patient inclines to the diseased side.

The thoracic vibrations are increased and respiration is reinforced over the area of hepatization. It then increases in frequency, the bronchial souffle is replaced by a tubal souffle and beside the sibilant râles of bronchitis crepitant râles appear with their ordinary characters.

But it is rare for actinomycosis of the lungs to affect the aspect of lobar pneumonia; it is a pseudo-lobar pneumonia that occurs. Therefore, the stethoscopic signs are very varied and mixed in the same patient.

In both the forms I have just described these signs persist during the phase of induration and hepatization of the pulmonary paren-

chyma, but then as we know from the pathology of the process that small and large miliary abscesses form in the midst of the lung. These abscesses undergo caseation and develop into cavities, and then signs common to pulmonary suffering are met with. Beside areas of dullness one will find areas of tympany. The nutrition is greatly involved, emaciation increases and the appetite diminishes. Large moist râles replace the crepitant or subcrepitant dry râles, the amphoric-cavernous souffle succeeded the bronchial souffles. The cavity then assumes the form of a pulmonary abscess with a hyperplastic connective tissue wall surrounding it. But this abscess not only invades the lung and extends to the adjacent organs, the diaphragm, pericardium posterior mediastinum, vertebræ, etc.—so that this is still another differential sign from pulmonary tuberculosis.

The actinomyces extend to the pleura, set up a very fibrinous pleuresy that the associated bacteria transform into an empyema, and from here the process extends to the thoracic walls giving rise to sinuses. The pulmonary actinomycosis has become a thoracic actinomycosis. This evolution and transformation are the almost necessary outcome of the pulmonary lesions, so that pneumo-actinomycosis becomes a pleuro-pneumo-actinomycosis which I shall now consider.

*Pleuro-pulmonary Form.*—The invasion of the pleura by the actinomyces often follows a focus or abscess in the lung which empties into the pleural cavity, but a pleural empyema has been known to follow an actinomycosis of the skin of the thorax.

In the majority of cases that have been verified by exploratory puncture, the pleural fluid was serofibrinous at first and resulted from the irritative process in the lung caused by the fungus. In Hausser's case, the fluid was clear and lemon-colored but did not contain the actinomyces. In cases observed by Dor, Bérard and Netter the fluid had the same characters while in Henck's case it was cloudy, but it is to be remarked that puncture was done quite a few days after the appearance of the symptoms of pleuresy, so that it is more than probable that the fluid had already undergone changes from bacterial association.

Independently of the general symptoms and the onset as described the pleural collection is diagnosed by the usual signs of pleuresy, namely, a pain in the chest, complete dullness on percussion, and absence of vesicular murmur, while souffles, friction sounds, râles,



pectoriloquy and egophony may be met with but offer nothing that is pathognomonic.

When once the actinomyces have entered the pleural cavity they continue their work. They invade the walls of the thorax which become hard, cedematous and painful. Then a tumor develops. Occasionally a kind of dense thoracic plastron develops. The skin is tense, red, painful and keeps the imprint of the fingers. The tumor, no matter what form it may assume, softens and becomes fluctuating; sinuses develop, giving exit to pus containing the characteristic grains, and communicate with a focus in the lung, and this brings one to the complications to which pulmonary actinomycosis gives rise. All that need be said, as might be supposed, is that all the adjacent structures may be involved in the process and that this extension takes place by continuity of metastasis. The parasite may break through the diaphragm and reach the liver or spleen, these viscera being previously adherent to the diaphragm, or extend to the renal region and, as has been noted by certain observers, form intra-muscular or subcutaneous abscess as far down as the thigh, this representing the so-called hygienic type of actinomycosis. In other cases the invasion of the parasite took place in the posterior mediastinum and spine.

*Differential diagnosis.*—As I have pointed out, pulmonary actinomycosis has no really characteristic clinical signs. From its varied forms it assumes the characters of numerous pulmonary affections, one might even say all, and this is especially true when the case is seen near the onset.

Following a bronchitis or a neglected cold, the patient begins to lose flesh, appetite and strength, has nocturnal sweats and on auscultation signs of softening or even of a cavity are detected, the clinical diagnosis of tuberculosis is made. In fact, the facial expression is that of pulmonary tuberculosis, with the flustered cheeks, dyspnoea and oppression. On the other hand, there is pain in the chest, the dyspnoea is not intense, while there is very moderate pyrexia or none at all. There are no hemoptyses and no Koch's bacilli in the sputum. The clinician hesitates and considers the question of pulmonary syphilis or pneumoconiosis.

The mildness of the inflammatory phenomena lead to the suspicion of sclerosis of the lung and in reality, this is exact, but the

etiological diagnosis is not made. The case is one of broncho-pneumonia or chronic pneumonia but the actinomycosis origin should be added.

The really interesting point in the diagnosis, and useful from the viewpoint of treatment as well, is the discovery of the actinomyces. In all obscure cases the foremost rule should be to recall the possible existence of actinomycosis. Of the affections with which this affection may be confounded. I shall not refer to tuberculosis as this disease is too well-known to require any comment, but will begin with pulmonary syphilis.

*Syphilis of the lungs* presents lesions quite similar to those of actinomycosis. Like the latter, it transforms the lungs into a sclerous tissue, giving the macroscopical aspect of sarcoma, while besides, there are areas of frank inflammation as well as caseation. It can be readily conceived how difficult the diagnosis may be in the circumstances, all the more so because the Wassermann may be negative and all external manifestations of lues may be wanting. But an enlargement of regional lymph-nodes would be in favor of syphilis since adenitis is absent in actinomycosis.

In pulmonary syphilis hemoptysis occurs because the blood-vessels are pathologically involved, the contrary being true of actinomycosis. But examination of the sputum is the most important of all. Let me add that this differential diagnosis is still more complicated by the fact that the exhibition of KI in large doses works well in both processes.

*Pulmonary Gangrene.*—Actinomycosis may be differentiated from gangrene of the lungs by the fearful fetidity of the sputum in the latter affection, expectoration in which pus cells, large cells infiltrated with fatty granulations, minute lumps composed by connective and elastic tissues and pigment granulations are to be found, all of which are wanting in the sputum of actinomycosis.

Pulmonary gangrene, whether circumscribed or diffuse, produces cavities whose walls have a special structure and whose centre communicates with a dilated bronchial tube. Around the latter will be seen a layer composed of pulmonary tissue detritus, elastic fibres and occluded vessels; a second layer presenting lesions of red and gray hepatization, and lastly a third stratum connected with the healthy pulmonary tissue by means of lesions of catarrhal pneumonia which

shade off into the normal tissue. Therefore, the aspect is quite different from that of actinomycosis of the lungs, while the former process offers special symptoms, namely, high pyrexia and marked adynamia.

*Bronchiectasis* will be differentiated from actinomycosis by the fetidity of the sputum and other signs but confusion with *cancer of the lung* may readily occur if care be not exercised, but in malignant pulmonary processes hemoptysis is frequent and offers the characteristic currant-jelly color. The sputum is composed of hematic, gelatinous sputum, containing elastic fibres and the cell elements of cancer.

The dyspnoea is paroxysmal due to compression of the pneumogastric and phrenic nerves. As in actinomycosis of the pleuropulmonary type there may be serofibrinous pleuresy but the fluid is more prone to be hematic, but these characters differentiate it sufficiently.

In *hydatid cyst of the lung* there are cough, pain, dyspnoea emaciation, hemoptysis and pleural complications resembling tuberculosis, but a vomitus will occur sooner or later in which vesicles or hydatid membranes will be found. In the absence of a vomitus, examination of the sputum will relieve all doubt.

In this rapid review of the differential diagnosis I have had pneumo-actinomycosis only in mind. The diagnosis becomes still more complicated when the pleurae become involved. I shall not refer to the diagnosis of these complications—abscess of the thoracic walls and axillary abscess communicating with pulmonary foci by sinuses, etc.—as these come under the head of thoracic actinomycosis and therefore do not concern us. For that matter, these complications greatly simplify the diagnosis as they at once indicate the nature of the process.

But when the pleura contains a serofibrinous or purulent collection the question may arise as to whether it is tuberculous, cancerous, syphilitic or actinomycotic. The clinical characters in themselves are insufficient for indicating the etiology of the affection so that the diagnosis will depend upon the sagacity of the physician, who should be suspicious of all morbid processes not clearly definite in their symptomatology and evolution. Exploratory punctures, followed by histological examination of the fluid will usually relieve all doubt.

The diagnosis of inflammatory affections of the lungs need not detain us. Whether there is bronchitis, broncho-pneumonia, pneumonia or pulmonary abscess, the only important point to be solved is their origin and nature. Briefly stated, regardless of certain data which differentiated pulmonary actinomycosis from the disease I have mentioned, there exist very great and intimate analogies which render the differential diagnosis a matter of considerable difficulty, but this may be made easier if only the physician will recall the possibility of infection by the actinomyces.

The prognosis of pulmonary actinomycosis is very serious, that of pleuro-pneumo-actinomycosis being especially so, and the only medical treatment, although very unsatisfactory, is the exhibition of KI in massive doses. The pleuro-thoracic forms of the disease enter into the domain of surgery.

# THE PATHOLOGY OF ACTINOMYCOSIS OF THE BREAST

By M. POITEAU, M.D.

From the Pathological Department of the University of Lille, France

HAVING consulted the recent literature on the pathology and histology of actinomycosis of the breast, I have come to the conclusion that I am able to add to the knowledge of the subject because the specimen was an unusually fit one for the study, the evolution of the lesions being sufficiently advanced so as to render possible certain really scientific and interesting verifications. My thanks are due to my friend, Dr. Lucien Féré for the four drawings illustrating the paper.

The breast, which was removed by Professor Duret from a woman, *æt.* thirty-two years, offered the following appearance: All around the nipple at distances varying from one centimetre to three fingers' breadth are seen a series of elevations over which the skin is thin and red. The skin presented ulcerations or apertures of various dimensions over these red areas, around which the skin was raised and from which pus escaped.

The pus was rather peculiar, being a dirty, grayish-yellow color, grumous and in places streaked with blood. Finally, it had a very special character. When examined microscopically it presented a very pronounced fatty infiltration of its component cell elements. This aspect of the pus is always suspicious of actinomycotic lesions and is a characteristic that should invariably draw attention, as it is yet one more element for making an early clinical diagnosis, because the yellow grains may appear in the pus only at a late date in the evolution of the process or on the other hand, may be very scarce. Now, if at the onset of the suppuration a microscopical examination is made, as it invariably should be, it is possible that the following characters of actinomycotic pus without the characteristic yellow grains, may be found: Besides the ordinary pus cells it contains bits of blood-vessels undergoing disintegration, connective tissue fibrillæ and detritus of various tissues, finally—a most important point—the leucocytes are, as I have said, infiltrated with quite a considerable number of fatty granulations.

It is clear that these findings are not enough to make a diagnosis of actinomycosis but nevertheless they give an indication as to the direction further investigation of the case should be carried out.

All the ulcerations in the specimen—some of which were the size of a silver twenty-cent piece—had thin, undermined edges; their fundus was granular and reddish and violet colored, granulations soft, extremely friable and bleeding upon the slightest contact, filled the loss of tissue. I repeat: All these sinuses and ulcerations were grouped in different parts of the surface of the breast in which areas the skin was raised, thin, red or violet and undermined and in these diseased areas the tissues were soft, depressible but without any very distinct fluctuation. The best description I can give is that, when pressed on, the lesions felt like putty, with this difference, that in the areas of ulceration or fungous products, a pitting from pressure with the finger did not remain on account of the elasticity of the tissues even morbidly changed.

At other parts palpation of the breast gave quite different sensations to the exploring finger. Portions of gland as hard as stone could be very well felt between the fingers and seemed to be well limited nodules of chronic mastitis. In one of the rare parts of the gland not yet involved by the morbid process a normal consistency of a portion of the glandular structure of the mamma could be indistinctly felt. Although healthy in appearance, it is probable that this had already undergone a certain degree of granulo-fatty degeneration or a generalized infiltration of its tissue by elements of sclerosis.

Finally, a most important detail, at no point on the cutaneous surface of the breast or perimammary region were cicatrices with a depressed centre to be seen. Nowhere among the numerous sinuses and ulcers did the fungous granulations present their characteristic aspect which likens them to tuberculous productions. The nipple and areola were absolutely intact.

The interior of the breast offers curious details concerning the topography of the lesions. Dividing the breast in different directions and making serial macroscopic sections in the areas of the ulceration, sinuses and elevations an important infiltration of the dermis was noted as well as a manifest thickening of the skin excepting, as might naturally be expected, around the ulcerations. Immediately under the skin, throughout almost the entire extent of the breast, a layer of actinomycotic tissue could be seen, a true uninterrupted sheet, so to speak, having infiltrated the subcutaneous cellular tissue. At certain spots this grayish, fibrillary, soft and friable layer became

thickened in a sort of neoplastic plaque which raised the skin. At all these spots the skin became gradually thin, and most of the cutaneous elevations noted at the external examination presented poorly limited irregular solutions of continuity; these represented the undermined festooned edges of the ulcers. Between the solutions of continuity in the skin neoplastic tissue projects upward giving rise to the plaques and subdermic layer of pathologic tissue referred to.

From these plaques prolongations shoot off and dip down into the midst of the gland, pushing in between the fat lobules and at length coming in contact with the glandular elements themselves and also penetrate their interstices. All these prolongations have the same structure as the subcutaneous neoformations, being made up by a yellowish-gray, fibrillary and extremely friable tissue whose component elements become disaggregated and ragged.

These prolongations starting from the superficial foci of disease, infiltrating through the fatty paramammary structures and glandular lobules themselves, usually end, after a more or less sinuous course, at other deep seated foci, some in the gland tissue, retro-mammary fat or in the pectoralis muscle itself. Very probably, if not to say certainly, these deep seated foci were of much more recent date because although some of them were large and presented an almost complete disintegration of the tissues by them invaded, there were others on the contrary—and these formed the majority—were not large or so well differentiated and had only invaded the gland at certain spots in a diffuse way, so that in spite of the infiltration it had undergone the glandular tissue still retained its normal characters almost completely.

At the areas of cutaneous ulceration and along the entire course of the sinuses, the tissues invaded by the parasite had actually melted away—a complete disaggregation of their component elements having taken place. They furnished a slow progressive elimination which was effected through the various strata of the mamma, giving rise to sinuses, and through the skin after the latter had undergone an inflammatory reaction, resulting in the production of ulcerative processes. It was at the oldest ulcerations that the skin was the thinnest, the foci softening the most extensive and the penetration of the lesions into the gland itself was the deepest.

Fig. 1 represents one of the serial macroscopic sections and, so to

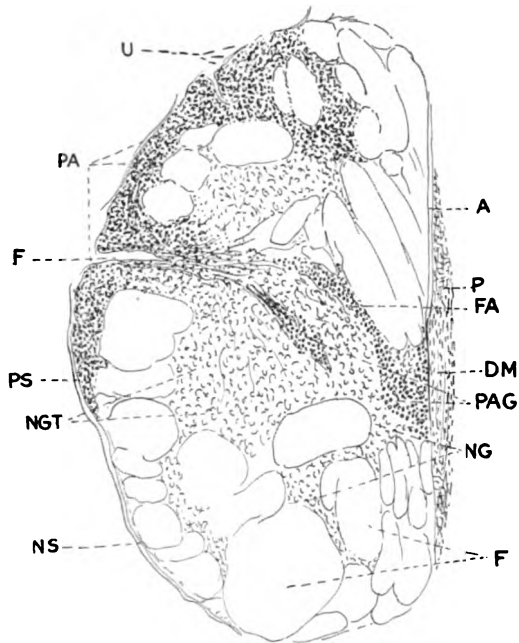
speak, depicts the foregoing remarks. It is a complete section of the breast passing just to the inner side of the areola where an old ulceration and a sinus opening existed. This section also shows a distinctly actinomycotic portion of the mamma, as well as an uninvolved area so that comparison is easy. The marked difference between the infiltrated right side and the relatively normal left side can be distinctly made out. There is nothing special to be noted here and one sees distinctly, going from the periphery to the depths, all the constituent parts of the mammary gland: The normal skin (N S), the anti and retromammary fatty layers (F), between which prolongations normal gland tissue dip (N G); and still deeper is seen the aponeurosis (A) of the pectoralis and finally the muscle itself (P).

In the right side of the section these various structures have, on the contrary, undergone important morbid changes due to actinomycotic infiltration. Thus, going from the periphery downward the following verifications can be made: The skin (P S) is thin, infiltrated, raised up and detached from the subjacent structures by the inflammatory actinomycotic process. The skin of all this region is not only thin, violet-colored and raised, but is also ulcerated at several spots, and presents some sinuses. Thus at F exists the external orifice of one of these numerous sinuses which burrow through the breast. The sinus is not seen in its entire length because it is oblique in relation to the section. A little more to the right at U is a very large ulceration and toward its middle can be seen a small sinus dipping down into the tissues.

It is easy to understand the process of invasion of the gland in this particular case. It takes place by both contiguity and continuity of the tissues; according to the data obtained from the patient, the actinomycotic degeneration occurring with the first abscess and consecutive ulcerations of the spernal region, penetrated the breast by working progressively under the skin and detaching it; then after having reached the areolar region which it respected, the actinomycotic degeneration turned its invasion to the deeper structures. It extended in between the para-mammary fat lobes and lobules while deep down the actinomycotic process (P A) has penetrated the interstices between the glandular lobes themselves, following the connective tissue fasciculi which serve them as a support and here and

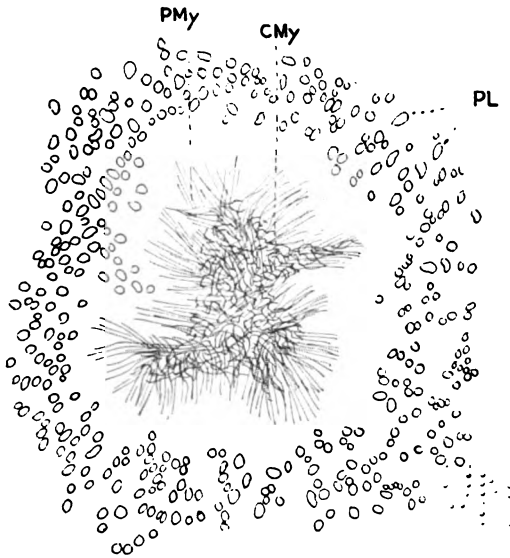


FIG. 1.



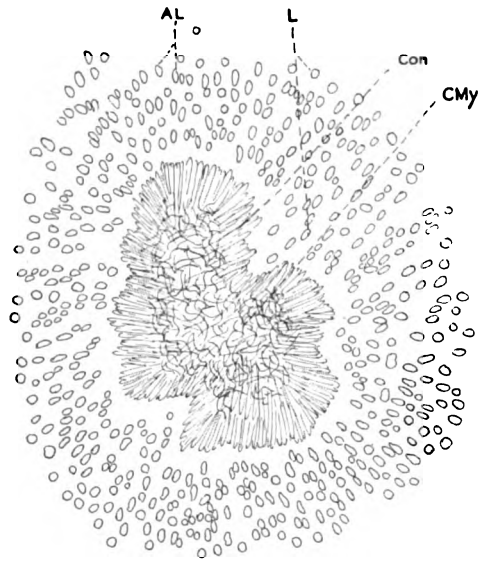
Section of breast enlarged twice its actual size.

FIG. 2.



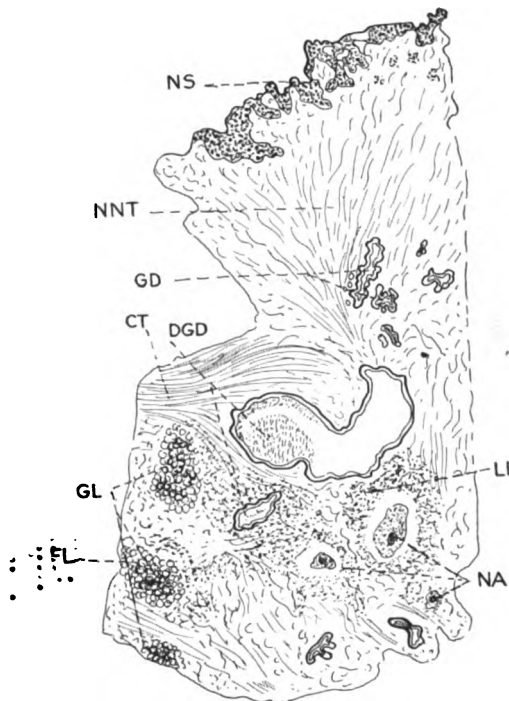
Young nodule (x860). CMY. Central mycelium. PMY. Peripheral mycelium which will produce conidia. PL. Polynuclear leucocytes.

FIG. 3.



Old node (x860). CMy, Central mycelium. Con, Conidia. L, Leucocytes in disordered arrangement. AL, Leucocytes arranged in order.

FIG. 4.



Section of the mammary glands including the nipple. (x50).

there form kinds of nodules, foci of degeneration, such as can be seen at P A G. At other spots, instead of these glandular or retromammary foci, the progressive infiltration, followed by degeneration of the infiltrated structures, has produced at F A true sinuses which contributed to the very rapid invasion of the gland at first and afterward the retromammary cellulo-adipose tissue, aponeurosis of the pectoralis and the muscle itself.

Fig. 1 shows, in fact, at D M, that a large portion of the muscle is undergoing degeneration; no normal muscular fibres can be seen, as for example at P. Above and within the same limits the aponeurosis (which is normal at A) can no longer be detected and at this spot there is a mass of yellowish substance which becomes stringy when drawn up with a needle or otherwise.

Microscopic sections were made in several parts of the gland. Some were made through the skin and subjacent tissues at spots corresponding to the ulcerations or sinuses. Other sections included the nipple and the gland tissue immediately subjacent. By inclusion in celloidin, which prevented the issue of sections of the actinomycotic grains, this second series of sections presented a larger surface than those of the first series. Therefore I preferred to take one of the former as a model and which is depicted in Fig. 4. All the sections were stained with iodine-hematoxylin.

*First series, comprising the ulcerated skin and subjacent tissues.*—These sections will serve to show the changes arising in the vitality and the condition of the structures invaded by the parasitic process and at the same time I will study the organization of the actinomycotic nodules in these infiltrated tissues. In each one of the serial sections the normal skin becomes thinner and finally disappears at the edges of the ulcerations and in its place granulation tissue occurs. This is composed of vessels with embryonal walls and dilated cavities, between which polynuclear leucocytes in quite large numbers or elements of young connective tissue can be seen, having the fusiform or stellate shape.

In going over the cutaneous surface in the section, it was found that the dermis presented very marked lesions of œdema. The fasciculi of connective tissue were dissociated and in the resulting network either leucocytes or connective tissue elements were seen, some being fusiform or stellate, others having a vesicular aspect. In those places

where the cell elements were less numerous, the connective tissue was infiltrated by a serous fluid which had become precipitated in the granular or filamentous form. There were even large lacunæ in which this serous precipitate assumed an important part in the evolution of the lesions. In point of fact, at these spots the presence of red blood corpuscles was noted scattered about irregularly in the interstices of the tissues and infiltrating them.

If now, the lesion of actinomycosis itself is sought for in these serial sections the following was to be found: Either in the almost immediate neighborhood of that portion of the section deprived of epithelial covering or in some deeper parts, stained masses in the form of rounded or polycyclical grains could be seen. From their almost violet stain these masses stood out wonderfully well against the surrounding tissue, from which, for that matter, they were quite distinct.

As has been said, all the sections were stained with iodine-hematoxylin according to Hauser's formula, because according to Professor Angier, this is by far the best for research work in tissues with actinomycotic lesions; therefore there is little advantage to be gained by using other stains. Iodine-hematoxylin has the advantage of differentiating and bringing out the actinomycotic nodule by imparting a special tint to the parasitic centre. For this reason I desire to briefly describe the preparation of the stain and its technic.

To 1000 grammes of a saturated ammonical alum solution, ten grammes of crystallized hematoxylin dissolved in one hundred quarts of alcohol, is added. On the other hand, a strong iodo-iodine solution is made as follows:

Distilled water .....	100 grammes.
KI .....	10 grammes.
Iodine .....	5 grammes.

Twenty grammes of which are added to the alum-hematoxylin solution. By boiling, maturation of the mixture is instantaneous or will be so in a few hours when left at room temperature. On the next day it is ready for use. It is better to dilute it with one or two parts distilled water in order to obtain a  $\frac{1}{3}$  per cent. solution which is the normal for all hematoxylin tinctures. The solution thus made up keeps well in a glass-stoppered bottle.

The sections are immersed in the solution for a few seconds to a few minutes, according to the age of the solution. Then, after having

passed the sections rapidly through distilled water to remove the excess of the stain the sections are then properly toned in tap water. The sections which are stained an intense red after immersion in the solution become immediately a uniform blue.

This technic is excellent and rapid for the search for actinomycotic nodules, but it does not bring out the exact arrangement of the filaments and conidia forming the centre of the nodule, so that when it is desired to examine the precise arrangement of the parasite within the nodule it is preferable to employ the following procedure devised by Doctor Angier.

First, the sections are placed in a 1 per cent. watery solution of methyl violet for two to three minutes; secondly, wash rapidly in tap or distilled water; thirdly, place them in Lugol's solution for thirty seconds to one minute, and fourthly, decolorize in 95 per cent. alcohol or in a  $\frac{1}{3}$  acetone-alcohol solution. After waiting until the section has taken a gray or grayish-blue tint it is then placed in pure anilin oil and left there until it is completely transparent; after which it is dried and mounted. This is, in reality, a modification of Gram's procedure but by treating with anilin oil the excess of the stain is removed and the sections are cleared. This technic is excellent for revealing the contents of the nodule as it has an elective action on the latter, but the remainder of the section is not stained so distinctly as with the iodine-hematoxylin solution; each has its own definite purpose.

Now, what is an actinomycotic nodule? What are its constituent parts and the nature of these distinctly differentiated masses, whose violet centre is surrounded by a zone of inflammatory reaction? The elements forming the centre of the nodules are radiated. In favorable sections with a high power and a homogenous objective, it will be found that this radiated arrangement is due to conidia grouped around one or several centres. Around these masses, particularly in the well-differentiated sections of nodules, the presence of leucocytes forming a rather thick border will be noted. This is the general arrangement, the aspect of a nodule examined as a whole. But if the details of its make-up be attentively and separately examined, it will be perceived that the actinomycotic nodule changes in structure according to the phase of its evolution and consequently the young and the old nodules should be studied separately.

I have had drawn for this purpose two illustrations in the light chamber and rather highly magnified. One of the nodules, the oldest, was selected in the subcutaneous tissue which had undergone actinomycotic melting; the second and younger was found deep in the breast situated in the glandular tissue itself, which apparently had only been recently invaded, because it showed little evidence of an important structural change. There are two elements in a nodule that are to be separately examined, namely, the parasite itself, which forms the centre of the nodule and the inflammatory reaction of the ambient tissue undergoing change and which forms the periphery.

If therefore a young nodule be examined, such as is depicted in Fig. 2, the following details are to be remarked: Right in the centre is the parasite, represented by the mycelium undergoing evolution (MY). The mycelian elements, mixed together in a very irregular way in the centre, are very regularly arranged at the periphery. The peripheral portions of these filaments are arranged parallel to each other and give the first indication of a tendency to peripheral radiation which characterizes the adult grains of actinomycosis, only at this stage the conidia are not yet formed. The peripheral mycelian ends have not as yet undergone degenerative transformation.

All around and at a short distance from this parasitic mass the reaction of the living structures has commenced its evolution. The elements composing the tissue which shelters the parasite have disappeared and an important leucocytic infiltration has taken place, many polynuclear leucocytes (L) are grouped about without any apparent order, thus forming around the parasite a zone of defence destined to engulf it. During this time the parasite, which is in the centre of the tissue undergoing reaction, continues its evolution of degeneration. The inflammatory reaction in the living tissues progresses and soon a complete systemization in the production and grouping of the leucocytes at the periphery is seen.

Fig. 3 distinctly shows this process and transformation. Thus, around the central parasitic nucleus—which at present has assumed the characteristic aspect of a grain of actinomycosis—the polynuclear leucocytes, which were previously scattered about without any order, are now grouped in a special way. These elements form a rather thick and individualized border and assume a radiated arrangement

near the grain. The nuclei of these leucocytes are elongated; they appear to be more or less oval and arranged in the same direction as the prolongations of the conidia (C and A L). The most external elements of the wreath of cells surrounding the grain are, on the contrary, arranged without any regularity (L). But, nevertheless, as they are more heaped together than the nearest elements outside of the grain, they form a quite distinct border around the latter which individualizes it.

The cells composing the nodule as a whole have nearly all a deformed multilobate nucleus; these are the polynuclears. There is here, just as in tubercles, a grouping of cells that may be interpreted as a true positive chemiotaxis.

As far as the parasite itself is concerned, one remark is important. In point of fact, from what we know of the phases of evolution of the actinomyces in living tissue, it is evident that there exists a great analogy between the aspect of the adult parasite in the pus and that in which it presents itself in the midst of the tissue of inflammatory reaction in the interior of the nodule. In both, the conidia exist in very large numbers and the apparent differences—disappearance of the mycelium and fragmentation of the grains contained in the pus—are in fact merely due to the unfavorable conditions of nutrition to which the grain is submitted, from the fact that it has remained in the pus for a more or less considerable time and at a somewhat more advanced phase of degeneration.

*Second series of sections, comprising the nipple and a portion of the immediately adjacent mamma.*—One of the sections was drawn with a projecting apparatus at x50 and will serve to explain the details to be described (see Fig. 4).

When the patient was examined clinically, the nipple and areola appeared to be intact and, in reality, in this section the tissue of the nipple appears normal (N S) and is covered by ordinary skin. In its thickness fasciculi of unstriated muscular fibres can be perceived while the galactophorous ducts (G D) show their usual sinuosities without any inflammatory reaction of the interposed tissue. Then in a deeper stratum of the section lesions of actinomycosis appear with abundant proliferation and massing up of the leucocytes. Here and there some galactophorous ducts are visible and one of them has undergone an enormous cystic dilatation with desquamation of its

epithelium (D G D). A portion of this dilatation is filled with cell elements of lamellar appearance and granular detritus. At the periphery, these cells present a flattened nucleus and in attempting to ascertain their origin it was found that they resulted from desquamation of the most superficial elements of the pavement epithelium lining the lumen of the galactophorous ducts. There are very few pus cells in this cystic dilatation. Other galactophorous ducts situated in the midst of inflammatory tissue offer partial dilatations, some have a normal appearance.

Some glandular lobules are seen in the section and for the most part are degenerated (G L). The glandular elements composing them are close together as if bound tight by the infiltrated interlobar connective tissue. The lumen of the acini and mammary canaliculi has from this fact disappeared while the glandular cells themselves have slowly undergone granulo-fatty degeneration.

In certain deep parts of the section and at varying distances from the cystic galactophorous duct actinomycotic grains were also found, retained in the section, thanks to the preliminary inclusion in celloidin and are stained violet-garnet by iodine-hematoxylin. There are three actinomycotic nodules to be seen (N A), differing in size but having an identical structure.

To briefly sum up it can be said that the lesions of actinomycosis are not directly related to the acini and galactophorous ducts, neither do they penetrate the constituent elements of the gland. The connective tissue frame-work of the mamma only is infiltrated with leucocytes, especially noticable around the nodules (L I). Nevertheless, the gland itself, as soon as it is bound in by all these infiltrated tissues—whose purulent melting continues without retrocession—can no longer resist; its elements when once compressed, end by secondarily degenerating and are not long in disappearing.

A lymph-node removed from the axilla of the patient and examined histologically did not show any phenomenon of degeneration or parasitic infiltration, only a little inflammatory reaction of the lymphatic tissue.

In closing I would say that although a free and bold excision of the breast carried out *in healthy tissue* is absolutely necessary to effect a cure, any enlarged axillary lymph-nodes can be safely left *in situ* as they are never the seat of lesions of actinomycosis.



## THE CLINICAL ASPECTS OF ACTINOMYCOSIS OF THE BREAST

By S. MILEFF, M.D.

Breznik, Bulgaria

---

PRIMARY mammary actinomycosis, fairly common in the bovine race, is certainly rare in the human. In order that infection of the breast shall take place the subject must be directly in contact with cereals or diseased animals, or else the actinomyces must be transported in some way to the midst of the mammary tissue or galactophorous ducts.

These etiological conditions can be realized in farmers who work in the fields with their thorax exposed, but there are cases in which the mechanism of the infection remains a mystery. If in Ammentrop's case cereals may be incriminated since the patient remained a fortnight working in the fields with the chest exposed, other reported cases are not clear. MacArthur's patient seems to have been infected directly by way of the galactophorous ducts as usually occurs in animals since no other trace to the parasite could be found in this case.

All that can be said is that *primary mammary actinomycosis does occur in man, and that in animals the infection takes place through the galactophorous ducts—a fact experimentally proved by Johnes—and that this way of infection has been known to occur in man. In the latter, the grains of actinomycosis may penetrate the tissues of the breast through a solution of continuity of the integuments due to a traumatism or some cutaneous affection.*

By far the greater number of cases of actinomycosis of the breast are secondary, pulmonary actinomycosis being usually the primary focus of the process, but I have notes of two cases in which the mammary lesion was secondary respectively to pleuro-lumbar actinomycosis and mastoid infection by the parasite. The last case was a woman, *æt.* twenty-six years, who developed what looked like a boil over the left mastoid process. Yellow grains of the fungus were found in the pus. Temperature 105° F. There was a purulent col-

lection along the internal jugular down to the breast, with metastatic abscesses in the liver and kidneys. Death occurred in six days. This is an example of an unusually acute case running an extraordinarily rapid evolution.

I here append a drawing from life of a case of actinomycosis of the breast secondary to a pleuro-lumbar infection by the fungus. The patient's history is too long to report in detail, but suffice it to say that the lesion adhered *en masse* to the costal wall and looked much like a cancer *en cuirasse*. The skin was like that of an orange. The woody infiltration reached the axillary region and at this point the swelling of the breast ended in a hard ridge. The arm was applied to the trunk; movements of abduction were limited in extent (see Fig. 1). Pain was severe, the axillary lymph-nodes were not enlarged and a pyrexia was complete.

There was no fluctuation but finally the lower portion of the breast became violet and an enormous collection made its escape through seven or eight small sinuses in the submammary fold near the axilla and it was after this event that ulceration appeared on the skin (see Fig. 1). The pus examined on several occasions revealed the actinomyces. The patient's general health became poor. KI in large doses and other medical treatment had no effect on the process although continued for two months.

The patient was lost sight of for two months. She then returned and was found to be in really flourishing health. She did not suffer and could move the arm with ease. Most of the ulcers had cicatrized or at least appeared so and only those at the lower part of the breast still gave exit to pus. There were now very few yellow grains in the pus.

This cicatrization was however only apparent and the patient knew it full well, because every fortnight or so an inflammatory attack occurs with abundant discharge of pus from the sinuses, and then all subsides. The case is one for radical surgical interference which will have to be done sooner or later.

*Symptomatology.*—When actinomycosis of the breast develops as a primary lesion it presents frequently the symptomatology of either tuberculosis, syphilis or a malignant neoplasm. What is striking is the development of a hard, oblong tumor in the mammary gland.

At the onset the growth is practically painless to pressure; it

becomes rapidly indurated and may reach the size of a peach or large egg intimately adherent to and quite inseparable from the gland. The skin also becomes adherent and tense; but without change of color at first; however, as the lesion extends it becomes red. The axillary lymph-nodes may or may not be enlarged, but when they are the enlargement is due to a secondary infection and not to invasion by the fungus itself.

Finally, manifest signs of inflammation appear in the breast; the skin is hot, red and painful and becomes perforated by sinuses from which pus, containing the characteristic yellow grains, escapes. The sinuses become chronic, resembling those met with in tuberculosis of the mammary gland, having no tendency to close and giving exit to a small amount of pus occasionally mixed with blood. Other sinuses develop at other points as the process extends. If these sinuses be scrapped a certain resistance will be felt due to the development of fibrous tissue.

*Diagnosis and prognosis.*—The evolution of actinomycosis of the breast is one of invasion and extension and in most cases the tumor will have attained the fistulous phase within the space of two or three months. In some instances the evolution is slower, especially an extension to the surface.

When the process begins in the mammary parenchyma it may be assumed that it can extend to the thoracic walls and lung although I know of no reported case in which this took place.

The diagnosis is always a matter of difficulty and is never made until pus makes its exit containing the actinomyces, unless of course, the patient presents other localizations of the fungus. The sinus or sinuses resist all kinds of treatment, the hard connective tissue and granulations bleed freely and are with difficulty scrapped away. This fact should lead one to suspect actinomycosis.

Consequently, *the exaggerated development of connective tissue in the growth, the difficulty of scrapping away the granulation tissue and the marked hemorrhage following* are three excellent signs which should lead to the search for the actinomyces in the pus or products removed by the curette. And if yellow grains are not at once found, it is not to be concluded that the case is not actinomycosis, because this search is frequently fruitless at first and the fungus will be detected only after a number of examinations have been made. In

MacArthur's case microscopical examinations of a bit of tumor removed for this purpose only revealed normal tissue and it was only at a later date that the fungus was found in the pus. In one of Müller's cases nothing was found in the secretions and the actinomyces was only found microscopically in the tissues removed.

When the phase of suppuration has been reached the process may readily be mistaken for tuberculosis or syphilis. If the case is a pulmonary actinomycosis mistaken for pulmonary tuberculosis with extension to the mammary gland, the mistake is fatal because tuberculosis will only be thought of, and it is only by careful consideration of the patient antecedents that this diagnosis will be eliminated. An early developing sharp pain is decidedly in favor of actinomycosis.

When the tumor is adherent to the skin it may be mistaken for cancer because it forms one mass with the gland; the nipple is often retracted and the axillary lymph-nodes may be enlarged. As I have already said, this adenopathy is never due to the fungus itself, but to secondary bacterial infection.

Let me add that the pain, rapid evolution and more or less distant secondary nodules will also be the cause of diagnostic mistakes. Snow made a diagnosis of malignant neoplasm, the lesion having developed in seven months and the indurated mammary gland was adherent to the thorax, while a secondary nodule developed in the scapula. At autopsy a primary pulmonary focus of actinomycosis was found that had invaded the breast secondarily.

Ammentrop excised the breast and axillary lymph-nodes believing he was dealing with carcinoma of the breast, while confusion with mastitis is all the more easy because occasionally, a traumatism precedes the first manifestation of the process.

After spontaneous opening, the sinuses may be mistaken for those due to tuberculosis and this is all the more likely when microscopical examination of the pus does not reveal the actinomyces.

Finally, when the diagnosis is made the question arises as to whether or not the mammary lesion is primary or secondary to a pulmonary focus or if a more or less latent dental infection does not exist as Müller has pointed out.

The prognosis of primary actinomycosis of the breast is less serious than the secondary form coming from the lung. Although actinomycosis is a serious affection, rapidly infecting, an energetic

treatment may arrest its evolution. In the primary form the recovery should be 100 per cent.; on the other hand, in the secondary form out of a total of nine cases that I have collected from the literature, there were five deaths and in the remaining four the outcome was unknown.

*Treatment.*—Large doses of KI usually fail. Surgical interference will be necessary in all cases, the extent of the operative act being guided by the extent of the lesion. When the process in the breast is secondary to a focus in the lung. KI in large doses *per os* with proper antiseptic treatment of the lesions is about all that can be done.

# ACTINOMYCOSIS OF THE TONGUE, WITH ILLUSTRATIVE CASES

By DENIS BONNET, M.D.

Former Intern of the Hospitals of Lyons, France

---

ACTINOMYCOSIS of the tongue is unquestionably one of the rarest localizations of the parasite, and it is somewhat difficult to give an exact estimate of its frequency in relation to other manifestations of this morbid process. Ljungdren states that he has met with one case in the tongue out of seven cases of actinomycosis but I believe this percentage to be exaggerated when one considers the few cases that have been reported.

I believe the infrequency is due to two causes, namely, an anatomical and a histological cause. The tongue is protected by the double barrier formed by the lips and especially the gums and teeth, and consequently is relatively well protected from those foreign bodies which are usually the carriers of actinomycosis. On the other hand, it is admitted that the connective tissue is the preferred soil for the evolution of the germ, and in the tongue this tissue is present in very small amounts. The organ, protected by a thick mucosa and formed by a mass of tightly woven muscular fibres which are directly united with the mucosa without any intervening submucosa, offers a very unfavorable soil for the development of the parasite.

Actinomycosis is an affection of adult life, therefore during the phase of greatest activity when chances of contamination are the most numerous, but Brunner has met with a case in a girl, thirteen years old. Males are four times more frequently attacked than females, this being due to the nature of their respective occupations. Almost all the cases have been in peasants, coachmen, stable hands and those whose work bring them constantly in contact with vegetables; however, Brunner has collected seven cases of actinomycosis in city people in whom the vegetable origin seemed to be wanting.

Climate and season play an unquestionable part. Rainy years or damp seasons are the epochs during which the largest number of cases develop and it is well known that the actinomycoses develop

much better on damp cereals than on dry. Lenson observed an epidemic which broke out in a flock which was grazing on rye growing on marsh land. The geographical distribution of the disease has been regarded too exclusively, nevertheless all countries are not equally subject to the parasite. Among those paying the largest tribute are Austria, Germany, Switzerland and some parts of Russia, all being damp regions, and whose culture of cereals and cattle-raising represent the main occupation of the people.

The mechanism of the infection resides in the ease with which the actinomycoses live on vegetable matter. There are three manners of contamination, namely, (1) following a direct traumatism creating a solution of continuity and at the same time introducing the parasite; (2) following an infection of an already existing injury or a diseased tissue; (3) following a fortuitous introduction without effraction of the actinomyces into an organ. The first two are practically the only causes of actinomycosis of the tongue.

In the immense majority of cases the subject has been chewing a spear of wheat, oat or barley which excoriates the tongue and inoculates it. According to Bostrom, who has particularly insisted on contamination by cereals, the germ penetrates within the barley grains by the apertures, that he describes, which develop in the ripe, dried grain and this observer believes that there is no external sign by which the presence of the actinomyces within the grain can be suspected.

This way of infection has been demonstrated by Krautz and Tribout in African cattle; in the diseased animals they found prickles and vegetable hair bristling with actinomycotic buds and with elongated cavities containing the spore in the tongue. The most curious instance is that of von Heille, in which one hundred and forty-one grains of barley were found and which had sprouted in the mouth, pushing roots the length of one inch and a half into the tongue; the stems charged with chlorophyl forming a small reserve garden at the bottom of the buccal cavity.

In the following case reported by Turinka, of Gratz, histologic examination revealed the presence of excessively thin barley grains interwoven with a network of mycelian filaments which united them together and to the walls of the abscess.

*Case 1. (Turinka).*—Male, *æt.* thirty-six years, presented a swell-

ling in the tongue and stated that about four months previously he had noted a small induration in the left half of the organ, which he attributed to a self inflicted bite of the tongue two months before. A small nodule slowly formed and after a time gave rise to great pain in moving the tongue, especially during the act of eating. The general health and nutrition were perfect.

He stated that he sometimes chewed wheat but that he never felt any pain in the tongue when so doing. On the other hand, he smoked a pipe with the stem always in the left side of the mouth directed exactly toward the spot where the swelling later developed.

*Examination* showed on the left edge of the tongue about two centimetres back of the lip, a small tumor, distinctly fixed, circumscribed, rounded, hard and the size of a small cherry. It was covered by mucous membrane which was slightly livid, could not be displaced over the tumor but offered no histologic change. Other than for caries of three molars, the mouth offered no pathologic change. No enlarged regional lymph-nodes could be detected.

The tumor, hard, painful, slowly increasing, in a smoker of middle age and produced after a bite of the tongue made one suspect a can-croid developing in the deeper strata of the lingual mucosa but did not exclude the presence of actinomycosis. The neoplasm was excised in healthy tissue and the wound healed by first intention in a week.

The neoplasm was split by an incision carried vertically to the edge of the tongue and under the slightly thickened mucosa a sperical growth about eight millimetres in diameter, composed of a resistant whitish tissue with a large central cavity about three millimetres in diameter in which were fixed five little, rounded, light-gray grains, rather hard and the size of small oatmeal. Two were examined microscopically, the remaining three were used for cultures which remained sterile probably because of an insufficiency of material used.

The grain examined at + 50 in a diluted potassium solution was found to be made up of an agglomeration of cryptogamic foci shaped like a mulberry. Among them were three situated near the centre, each offering a cluster of conidia included in a pale-gray substance from which they well contrasted by the greenish-yellow color.

Examined at + 500, they were seen to be true actinomycosis in the shape of a typical grain. The isolated foci consisted of a central network of straight or irregular filaments with true arbori-



zations running off in every direction, which in many places were closely attached to these conidia. Between them numerous free formations, similar to cocci, were seen. The entire focus was surrounded by a zone of brilliant pear-shaped conidia which adhered to the radiating filaments.

When the central conidia were examined under a high power they were found to be greenish-yellow and measured from one-tenth to one-quarter of a millimetre long, more or less adherent to each other, and usually parallel. Some presented a narrow, lighter intermediary space, placed between two cell groups stained darker and parallel to each other. This was the appearance of the grain. In the area about almost all the conidiophores was an extensive development of the fungous process.

This manner of infection, as in the case just related, is not exclusively confined to the tongue but all observers have noted it. Fischer and Schartan have found these grains in actinomycosis of the tongue; Illich, Bostrom and Gravit, in actinomycosis of the jaws; Soltmann, Beatha and Bostrom, in the region of the neck; Illich and Bostrom, in abdominal actinomycosis, while Piana found them in three cases of actinomycosis in the cow, and Johnne found grains completely riddled with actinomyces in the tonsils of a pig.

Another argument in favor of the cereal origin of the disease is that only omnivorous animals are afflicted, it never having been encountered in the carnivorous. All fodder does not offer the same degree of contagion. The parasite occurs more frequently and is more active when little care is taken of the fodder. When the latter is heaped before complete drying has taken place, etc., it offers the most favorable conditions for the development of the fungus.

Besides this way of infection which is by far the most common, there are other cases in which the fungus comes from an animal, as in the following case.

*Case III. (Maydl).*—An employee, obliged to examine the passports of cattle at a station where the animals constantly transshipped, was in the habit of moistening his thumb on the back of his tongue. Some very painful rhagades developed, followed by an abscess the size of a pea, the pus containing actinomyces.

By passage through an animal the virulence of the fungus becomes attenuated. Consequently in Maydl's case, as Bérard has pointed out, as well as in those observed by Guder, Hartmann, Illich,

and Poncet, the parasite had preserved an exceptional virulence and also there exist malignant forms of actinomycosis in animals, just as they exist in man.

As to contagion from man to man, only two cases have been recorded but these were not cases of tongue infection.

It has been questioned whether or not feeding on infected vegetables or animals might not be the cause of contamination. This is an hypothesis that no data have as yet been able to prove, at least as far as the tongue is concerned, because Chiari's case of intestinal actinomycosis cannot be explained in any other way than by contaminated food.

Such are the determining causes of actinomycosis. Their action is favored by certain predisposing conditions which remain to be considered. In the first place all the traumata should be mentioned, not only those due to the agent bearing the fungus itself, but a traumatism received before the infection, which acts by preparing the soil by causing the tongue to be a *locus minoris resistentiae* in which the actinomyces can readily develop. Usually, they are erosions of the tongue produced by rough tooth stumps as in Turinka's patient. At other times it is a bite, a burn or an erosion produced by some foreign body.

But besides such manifest traumata, the irritating action of certain substances, especially such as tobacco, must not be overlooked. Turinka's patient stated that he was a confirmed smoker, holding the pipe so that the stem grazed the tongue always at the same spot where later the actinomycosis developed. On the other hand, Didsbury has recorded a case of laryngeal actinomycosis which this observer attributes to the use of cigarettes. It would be interesting to know if actinomycosis can become grafted on certain common lesions of the tongue, such as syphilis, tuberculosis, etc., but I am unaware that any such instance has been observed.

Finally, certain diathetic conditions, such as alcoholism, pregnancy, tuberculosis, typhoid fever and all other conditions having an unfavorable influence on the organism in general, such as defective feeding or the want of proper hygiene favor the development of actinomycosis for reasons obvious to all. Guérmonprez and Bécue mention a case of actinomycosis of the jaw in a young man fourteen years old, who completely neglected all care of the buccal

cavity and with many defective teeth, a condition rather unusual in so young a subject. Such a condition unquestionably favors the entrance of the fungus, or at least it would not remove it from the buccal cavity as the detritus of food is allowed to remain. Consequently, an essentially favorable medium is offered to ferments of all kinds on account of the many anfractuositities in the mouth, the moisture and warmth which exist and therefore make it an excellent soil for the implantation of the fungus. The latter having gained entrance can vegetate for some time until some solution of continuity allows it to enter the organism there to multiply and exercise its ravages.

Briefly put, lingual actinomycosis is due to the development of the fungus brought in contact with it, by some cereal in most cases; its development being favored by a state of tissue inferiority due to a traumatism or a diathetic state.

*Pathology.*—When they have penetrated the tissues, the actinomyces act as a foreign body. They provoke phenomena of reaction whose intensity varies according to the vitality of the tissues and the virulence of the parasite. It may happen that the fungus dies at the onset of the process but in most cases it is triumphant, producing a tissue irritation around its site, the first result of which is an active proliferation of the fixed cells of the interfascicular connective tissue of the tongue. This tissue becomes more and more indurable, undergoes sclerosis and increases in bulk pushing aside the muscular fibres which at length undergo atrophy. In this first phase of the process, in every way similar to the corresponding phase of tuberculosis, the lesion presents the aspect of a small connective tissue growth, hard and enmeshed among the adjacent atrophied muscular tissue. This is the actinomycoma.

The following is an account of a microscopical examination that I carried out on a focus of lingual actinomycosis in an ox, at the veterinary school at Lyons. The section was made in a transversal plane perpendicular to the long axis of the organ in the midst of the tissues comprising actinomycotic lesions and remote from the mucosa. The muscular structures, at a point rather far removed from the parasite, were still healthy, grouped in distinctly limited fasciculi; having kept their distinct striation and their brownish-red stain to picrocarmin. But nevertheless, what can already be noted

at this point is that the fibrous bands are very thick and that as the nodule occupied by the fungus is approached the connective tissue offers a less marked pink stain. Points of granular degeneration can be detected. The blood-vessels at the same spot present a notable thickening of their connective-tissue wall, but without proliferation of their endothelium.

As the parasite is approached, the muscular fibres piled up eccentrically lose their striation and have undergone a vitreo-granular degeneration while at the same time their stain recalls more that of the pink tint of the connective tissue. They can then only be recognized by their fatty glitter which stands out against the duller background of the preparation. At this spot the vessels have almost completely disappeared.

When the parasite is still more approached one comes to a zone occupied almost exclusively by cells, some of which still resemble leucocytes; the more central ones in relation to the lesion having undergone various morbid changes. This is the actinomycotic nodule which is only slightly limited at the periphery by the sinking down of the connective tissue and muscular fibres.

Starting from the periphery, the first cells met with are almost normal leucocytes, but whose clusters are, however, already strewed with minute granulations derived from the destruction of deeper-seated cells. The latter, in almost direct contact with the yellow grain, have for the most part preserved distinct contours, but some have the look of the large epithelioid cells of nodules of tuberculosis while others—and these are in the majority—have undergone the peculiar type of degeneration first mentioned by Unna as characteristic of actinomycosis. In the latter type of cell the nucleus can hardly be seen, being poorly stained and having little refraction. The body of the cell, very much spread out, shows a protoplasm limited at the periphery by a rather clear border, while as the nucleus is approached it becomes more cloudy and granular although still taking the stain badly.

Finally the parasite itself, whose groupings are multiple and with an absolutely irregular arrangement in relation to the centre of the general nodule, is seen in the sections stained with picrocarmin with its absolutely characteristic radiated border and its golden-yellow (picric acid) stain. No giant cell can be found around it,

showing that the lesion is a young one, and the degenerated granular or epithelioid cells come in contact in some spots with the pear-shaped conidia which seem to be fitted into their interstices without the interposition of a granular layer.

This arrangement, the intimate relationship of the parasite to the adjacent cells, explains its preservation in the sections, but this is not always the case. In reality, when several of these elementary actinomycotic nodules are examined it will be found that in some of them there are still some yellow grains but that in the majority the parasite is absent and the centre of the nodule presents a vacuola which was filled by the parasite in the fresh specimen. During manipulations the fungus will disappear all the more readily because between it and the degenerated cells of the immediate vicinity there is a zone of granular cells that diminishes the intimacy of the relationship between the actinomyces and these degenerated cells, a zone that can easily be distinguished in the preparations.

In becoming sclerosed the connective tissue acts not only on the muscular fibres but also on the vessels supplying the parts, so that the little tumor becoming deprived of blood undergoes a peculiar type of degeneration. A small amount of fluid results, rather more of a serous than purulent nature, in which the characteristic yellow grains of the actinomycosis lesions float.

These grains, which at first were pale gray, soon take on a beautiful bright-yellow color, which in turn becomes more and more dark and when this has occurred the exudate holding in suspension a large number of brown and sometimes blackish grains gives to the eye the illusion of an old blood focus.

While this disintegration is going on in the centre of the lesion, the parasites on its borders continue their work and invade progressively the adjacent tissues until a new element—bacteria—puts in an appearance and chokes the actinomyces, taking their place and giving a new evolution to the process.

In the normal state, the buccal cavity contains a large number of pathogenic bacteria, whose nefarious action, as far as the tongue is concerned, is prevented by the vitality of the tissues and the integrity of its mucosa. In subjects with actinomycosis, hygiene of the mouth has generally been sadly neglected, so that the buccal cavity is prone to contain a greater number of more virulent bac-

teria than are found in a well kept mouth. These bacteria will profit by any erosion that has served as an entrance to the actinomyces and will penetrate into the focus of the lesion.

Since the actinomyces cannot live in company with the bacteria of suppuration they give way and rapidly disappear, with the natural result that an ordinary abscess remains, containing grumous pus and walled off by an indurated zone, itself ready to undergo purulent melting. If at this time a surgical interference is resorted to, the surgeon will come upon a focus whose parasite will have completely vanished and if he be not in possession of precise data relating to the onset and evolution of the process, it will be impossible, or at least difficult, to make a diagnosis.

These secondary infections, frequent in man, are on the contrary very rare in animals. The explanation for this is only hypothetical. Is it because the pathogenic bacteria are less numerous in their buccal cavities than in man? Or is it because the lesion being more rapidly apparent in the latter, treatment is resorted to sooner?

It may for that matter happen that in man the process will evolve without the addition of the bacterial element. The actinomyces retains its vitality and reacts upon the adjacent tissues, causing the lesion to extend at the periphery, in every way similar to what takes place in tuberculosis.

The parasite may even enter the circulation and be transported to the viscera—especially the brain and lungs—where it forms new colonies. It is to be noted that the lymphatics having a calibre sufficiently large for the reception of the fungus are never invaded by it. Adenitis or lymphangitis is unknown. The patient I observed in Professor Poncet's clinic certainly did present an indurated submaxillary lymph-node, but she stated that it had always been there.

*Case III.*—Female, *æt.* fifty years, never pregnant and in excellent health, gave the following history. The present disease began in January. The patient said that she bit her tongue but no tooth was found which would have been capable of producing an erosion. At present there is a hard tumor in the left side of the floor of the mouth, completely submucous and occupying the depths of the middle linguo-gingival sulcus. No pain, ulceration or secre-

tions. This nodule was the size of a large almond, uniformly hard but having a peculiar elasticity.

The patient had been treated with local applications of iodine glycerine and it would seem that the tumor had decreased from their use, but it occasionally offered a larger size. The tongue was sometimes so tumefied—especially in the morning—that the organ almost completely filled the buccal cavity. There was a small hard submaxillary lymph-node which the patient said had always been there.

A few months before, this woman had taken care of some little pigs who had abscesses in the neck and she herself anointed them. From time to time she chews a bit of straw or cereal. There was no trace of any buccal ulcer or morbid secretion. She was ordered iodide treatment.

*May 12.*—The tumor had ruptured about three weeks ago and since this occurred one or two drops of pus made their exit each morning. The tumor had decreased considerably, but there was still some deep-seated induration in the buccal floor. No pain, ulcer or appreciable sinus. Sometimes there is a slight discharge of blood but no longer pus. A little fungous matter was scraped away and examined by Doctor Dor, who only found the characters of epithelium but no actinomyces. The patient was heard from in November of the same year at which time she wrote that no trace of the affection remained.

Let me say at once that the explanation of the extension of the morbid process to distant parts is merely hypothetical at least so far as the tongue is concerned. The lesions here are too superficial and apparent to be overlooked for any length of time and they produce functional disturbances of mastication—even when they are not painful—quite sufficient to call attention to the tongue at the onset of the process.

*Symptomatology.*—I have pointed out that there are three phases in the development of lingual actinomycosis, namely, the actinomycoma, softening and yellow grains and lastly, secondary infections. All three phases are to be found in the symptomatology but with much less distinctness than in the pathology of the process. The various symptoms of lingual actinomycosis are wanting in precision and vary as much from one case to another as in the different phases

of the process. On account of this I shall divide the symptoms into physical and functional.

The onset of the process is ordinarily quite distinct on account of the exquisite sensitiveness of the tongue and also because of the ease with which the organ can be examined. The patient himself connects the onset with the initial lesion—the erosion of the mucosa produced by a spear of oat, etc. Then following the excoriation he will note that the tongue becomes thicker, loses its mobility and is the seat of some tumefaction usually more evident at the site of the excoriation. In Case III, the patient mentioned the fact that the tongue was sometimes swollen in the morning while the following patient complained of it.

*Case IV. (Fischer).*—Male, *æt.* twenty-nine years, chewed a spear of barley, a bit of which penetrated the tip of the tongue and became implanted in it. The tongue swelled at this point and finally a tumor the size of a filbert, manifestly fluctuating, developed. On the under surface of the organ three small nodules the size of a millet seed could be detected. The tumor was painless.

The remainder of the tongue was normal and the patient would not have perceived anything if an abscess had not developed, the pus of which contained the actinomyces. The breath was very fetid. The mouth was disinfected and the abscess incised, healing taking place quickly. Search for the actinomyces made on some samples of barley coming from the same source remained negative. The patient never took care of animals.

After some little time small, hard nodules appear and are at first so deeply seated that their surface is on the level with the rest of the tongue. Then they enlarge slowly, pushing up the mucosa covering them and therefore becoming distinctly circumscribed.

In man there is often only a single nodule while in animals there are frequently several. Occasionally the tongue may be riddled with them and acquires a consistency, stiffness and an aspect that veterinary surgeons know by the name of “wooden tongue.”

The nodules are usually distinctly limited from the adjacent structures, fixed and rather regularly rounded. At first they are simply indurated spots, more easily felt than seen, and at length acquire the size of a cherry or an almond. They are usually hard. Case III had a nodule the size of an almond which gave a peculiar



elastic feel to the finger, while in Case IV there was from the onset a fluctuating nodule.

The epithelium of the lingual mucosa may be perfectly normal in every way as in the following case, or it may be more or less changed in appearance, the epithelium covering the nodule being somewhat livid.

*Case V.*—Male, *æt.* thirty years. No pathologic antecedents. Two months before coming under treatment the patient had noticed a small nodule projecting rather in the direction of the floor of the mouth in the right edge of the tongue not far from the tip. He attributed the lesion to an erosion produced a few months before from an upper first right molar. Some carious teeth had been removed at the time.

The small nodule at first grew slowly but a week before coming under observation it had attained considerable size although it was painless and did not interfere with the functions of the tongue. An exploratory puncture showed that the process was actinomycosis.

The patient was a farm-hand and had taken care of a cow that had a tumor the size of a fist on one side of the lower jaw which ruptured spontaneously giving exit to pus.

Examination of the patient's mouth showed that the teeth were in good condition and that the first right upper molar and last lower molar were wanting. Several small erosions were seen on the surface of the tongue, probably due to mastication of barley or oat grains.

The lingual tumor was the size of a cherry, covered by a normal mucosa and distinctly limited from the surrounding structures. No jaw lesions. In the centre of the tumor a yellow mass was found the size of a pea which microscopically proved to be actinomycosis. The patient was discharged, cured, a few days later.

In all the cases where a note was made of the condition of the adjacent organs, especially the remainder of the tongue as in Case IV, they were found absolutely normal. However, occasionally the aspect of the tongue is different and in Case II the lesions were constituted by rhagades and small fungous erosions in which small miliary abscesses were seen.

Moreover, in typical cases, if no treatment is instituted, the nodule softens, then becomes painful, the mucosa becomes red, thin and ulcerates, thus opening the little cavity from which a caseous

mass makes its exit containing the yellow grains, usually in a much altered state and mixed with pus. The bacteria of suppuration at first mingle with the fungus and in the end become substituted for the actinomyces. These abscesses have no tendency to heal and persist for a long time in the form of sinuses giving issue to badly mixed pus.

The functional disturbances do not have the same fixity as the physical symptoms. In some cases the pain is completely *nil*, even during mastication or upon pressure. In other cases, on the contrary, there is considerable sensibility as in Case I. When it exists the pain is exaggerated by movements of deglutition and mastication. It is uncommon, however, not to find some trouble in the accomplishment of the various functions of the tongue. However, in Case V, such functional disturbances were wanting, but this is an exceptional case and almost all observers have noted more or less marked stiffness, making movements of the tongue more difficult. It would seem that animals are exposed to loss of mobility of the tongue much more than man, and in the notes of two cases of lingual actinomycosis, one in a horse, the other in an ox, in my possession, I note that the veterinary surgeons, Doctors Truchon and Gibbings, insist upon disturbances in mastication and deglutition. It would appear that this is due to the greater frequency of numerous nodules in the tongue of animals than in man.

The breath is sometimes fetid (Case IV) but this symptom may very well be due to some other cause than the actinomycosis. Doctor Truchon noted considerable salivation in the horse.

In ordinary cases there is no disturbance of nutrition, and the general physical condition is undisturbed. It is only in untreated cases in which the parasite has completely invaded the tongue that life may be endangered, but this state of affairs has, so far only, been met with in animals.

Lastly, the evolution of the process is essentially chronic, at least this is true of the majority of cases. It is occasionally interrupted by acute outbursts of the process arising without any apparent cause as occurred in Case IV. Briefly stated, the process consists of more or less numerous nodules, varying in size, at first hard and softer later on, interfering with mastication and deglutition and frequently painful. Such are the clinical signs presented before incision and

microscopical examination of the morbid products removes all doubt as to the true nature of the affection.

The physician may be called upon to make the diagnosis of lingual actinomycosis at one of two phases of its evolution, namely, before incision of the tumor, when only nodules are present, or after surgical or spontaneous openings of the morbid focus.

Before opening, the presence of hard, slightly elastic nodules having a slow progressive evolution, painful or otherwise and without concomittant adenitis should, in absence of any functional disturbance of the tongue, lead one to suspect actinomycosis. At this phase in its evolution the process can hardly be confounded with any lesion other than syphilis or tuberculosis or perhaps with a neoplasm still deeply seated in the organ.

If the lesion be syphilitic, the patient's history, the Wassermann reaction, the presence of other luetic stigmata and especially induration of the submaxillary lymph-nodes will make the diagnosis probable, but it is to be remembered that KI is likewise a test treatment for actinomycosis.

If, on the contrary, the patient gives hereditary or personal antecedents of scrofulo-tuberculosis, if KI does not produce a notable improvement and if submaxillary adenitis is present, a tuberculous lesion must be suspected.

Non-malignant neoplasms are very uncommon in the tongue and of course KI is without effect upon them. Malignant growths rarely develop before the age of forty years, and epoch when actinomycosis is at least very rare. Moreover, as in syphilis and tuberculosis, epithelioma reacts rather rapidly on the regional lymph-nodes.

All things considered at this phase actinomycosis does not possess a single clinical sign belonging to it so that a sure diagnosis can not be made. This can only be done by considering the probabilities, these themselves being based on the antecedents, age and above all the submaxillary lymph-node involvement.

In animals, the question of diagnosis becomes complicated by still another cause for mistakes, namely the pseudo "wooden tongue," which has never so far been encountered in man, which does not mean that he is exempt from it, because all peculiarities of actinomycosis were first met with in animals before they were described in man.

Two varieties of lingual pseudo-actinomycosis have been described. (1) The first form, the most frequent, involves almost all of the anterior, half of the tongue which is at first tumefied and hard on the surface, then throughout its entire thickness. It never extends to the posterior half of the organ and there is never any history of wounding the tongue. The organ becomes harder and harder and if KI is not exhibited in massive doses death soon ensues since the animal cannot feed. This form is especially prone to occur in young animals during the second dental eruption.

The second form, which is more rare, is only met with in adult animals. It particularly, but not exclusively, occupies the dorsal portion of the tongue. Small, very apparent, well-defined elevations are seen, hard to the feel and composed of connective tissue, and may attain the size of a hen's egg. They are covered by intact mucous membrane. The total volume of the tongue is little increased, but its mobility is considerably diminished. The animal is lively and strong, although thin, while in the first form of the process there is considerable loss of strength, shakey gait and an expression of pain and distress. Nevertheless, it is the most serious form, resists all treatment and kills quickly. Imminger states that pseudo-actinomycosis occurs in the ratio of three to seven with true actinomycosis of the tongue.

When the focus has opened, especially when incision has been resorted to, the diagnosis becomes easier from the presence of the yellow grains, but this verification is itself surrounded by certain difficulties; the grains may be overlooked when they exist or they may be taken for agglomerations of organized elements.

To avoid the first cause of error, it should be recalled that the grain is very variable in size and color and that it becomes altered very quickly. Therefore, it must be examined as soon as possible.

Leucine crystals described by Coppen Jones in the sputum, cancerous suppuration and purulent granulations may impose themselves for yellow grains of actinomycosis of the tongue. If the bits examined resist dissociation with the needle when placed on a glass slide and cannot be reduced to the state of mush or in mucous filaments it is practically certain that they are the yellow grains of the fungus but microscopic examination will relieve all doubts.

The question is more delicate when the specimen is a fungus

other than the actinomyces, such as the *aspergillus niger*, *leptothrix*, *cladothrix*, etc., as well as all the other usual inhabitants of the mouth and pharynx, whose interference is so important in the accidents of secondary infection. It is usually enough to recall that the actinomyces is the only fungus possessing pear-shaped conidia in order to distinguish it from other types which, for that matter, rarely play a part.

Dor has referred to another type of mycosis whose parasite and symptomatology much recall actinomycosis, but the mycelian filaments are thicker and there are no pear-shaped conidia. However, given the similarity of the evolution, Dor suspected that this might be a very differentiated variety of actinomycosis.

In reality, a very exact diagnosis between these two fungi has only a theoretical interest, because, clinically, the symptoms, progress and prognosis are the same.

When the morbid focus opens spontaneously, the diagnosis will be relatively easy because the result is a kind of ulcer with distinct edges, not undermined as in tuberculosis, nor punched out as in syphilis.

From what has been said, it is evident that the diagnosis of lingual actinomycosis is rather uncertain in all the phases of the infection, especially the early phase and with Poncet it may be said that the process is "a mixture of inflammatory and neoplastic lesions which frequently lead one to suspect scrofulo-tuberculosis, but especially give the impression of some disease that one has never encountered before."

The prognosis is benign when the affection is seen at the onset and at once treated with KI. Of all the localizations of actinomycosis, that in the tongue is unquestionably the most benign, and this precisely because the seat of the process does not allow it to be overlooked for any length of time.

## THE HEPATIC LOCALIZATIONS OF ACTINOMYCOSIS

By GEORGE ARIBAUD, M.D.

Lyons, France

---

THE majority of observers only mention actinomycosis of the liver incidentally in connection with other manifestations of infection by the actinomyces. In *Le Dentu and Debel's Traité de Chirurgie*, Brodier merely mentions the possibility of extension of the disease from the lungs to the liver through the diaphragm and from the intestine to the liver by either continuity or by the venous circulation. In *Bronardel and Gilbert's Traité de Médecine*, Ménétrier gives the question some attention, while Roger, in *Charcot and Bouchard's Traité de Médecine*, remarks that the liver may be primarily involved by the fungus.

Baumgarten states that the process may extend from the intestine to the liver while the intestinal lesions may themselves heal, leaving hardly any trace of their former presence. Choux says that among the abdominal viscera, the liver may be the seat of primary actinomycosis. Following an obscure period during which the patient complains of pain in the right hypochondrium, the liver will be found to be enlarged and presents one or several nodules on its surface. Constipation is the rule at this time. At the expiration of a variable length of time, œdema of the abdominal wall appears and the focus in the liver opens on the surface through numerous sinuses giving issue to serous or purulent fluid.

Choux also adds that secondary actinomycosis of the liver is one of the most frequent lesions found, the actinomyces being carried to the liver by the portal vein from the primary focus. The fungus thrives in the hepatic parenchyma with great ease as the hepatic gland realizes the conditions of anaërobiosis which are favorable for the culture of the parasite.

As is seen, Choux admits that extension to the liver takes place by the veins; he also believes that this may occur by continuity, and he says: "In either primary or secondary abdominal localizations, actinomycosis may produce intimate adhesions between the intestinal

coils and other abdominal viscera; it may involve the liver, tubes, ovaries, kidneys, bladder, etc." He does not admit extension by the lymphatic system, as he says: "Actinomycosis may produce metastatic abscesses and adenitides which are due to pathogenic bacteria that have invaded the organism at the same time as the fungus. The actinomyces is too large to pass through the lymphatics."

This opinion is maintained by Partsch who says: "If there is swelling of the lymphatics in actinomycosis, experience has shown that it is to be regarded as simply inflammatory and due to concomitant suppuration. Actinomycosis itself does not give rise to metastases in the lymph-nodes a remarkable fact for an infectious process to which little attention has been paid. This is why it is possible to permanently cure the disease if the primary focus can be destroyed."

Hugo Langstein says practically the same thing: "It is unquestioned that.....actinomycosis extends from tissue to tissue, and it is not likely that it follows the lymphatic vessels. The fungus seems to be too large (for the lumen of the lymphatics), and no authentic case of lymphatic infection by actinomycosis has been reported. If the lymph-nodes swell it is a mere concomitant phenomenon, and if suppuration takes place the actinomycoses are not found in the pus. On the other hand, extension by the blood-vessels is distinct."

The same observer says that actinomycosis of the lungs may extend to the abdomen by going through the diaphragm. He likewise admits extension of intestinal actinomycosis to the liver by continuity: "The fungus passes through the mucosa and soon sets up strong adhesions with the surrounding structures. In these adhesions large abscesses containing the actinomyces are found, or the intestinal loops are united by fleshy granulation tissue in which the fungus develops in little foci. Thus intestinal loops are adherent to each other and may be embedded in a mass of connective tissue. The gut may adhere to the liver, spleen, bladder and genital organs."

Langstein also refers to venous extension: "The progress of the affection is much more rapid if metastases arise. In the case of actinomycosis of the abdominal parietes, metastases will be found in the liver. Extension takes place by the portal vein," etc.

Koranyi, of Budapest, also admits extension of actinomycosis of

the intestine and lungs to the liver. According to him the process "coming from the posterior wall of the cæcum or ascending colon, attacks the retroperitoneal tissue by contouring the peritoneal covering of the large intestine. Large irregular abscesses form and invade the kidneys, liver," etc.

"There are many metastases in the territory of the portal vein; they may pass through the liver at an early date in the process, in which organ they show all stages of development."

The fungus may also invade the organism by way of the arterial circulation and this route has served for its extension to the liver in one of Israël's cases; he says: "The progress of extension of the fungus, starting from the primary pulmonary focus, as well as the manner of generalization throughout the organism, has been made evident by microscopic examination. In the first place, the lymphatics of the pulmonary lobe and the neoformed lymphatics in the pleural adhesions absorb the sporulæ of the fungus in the form of masses of little grains similar to micrococci. From the lymphatic system they pass into the circulation and are from here lost in organs, giving rise to metastases with the exception of the liver. . . . . In the liver, the process is different from that just described. Here a portal embolus takes place."

It will be seen that Israël, contrary to other observers, admits the possibility of extension by way of the lymphatics and the importance of this route is undeniable as has been demonstrated by Pawlowsky and Maksoutow in their work on phagocytosis in actinomycosis. Several of the illustrations accompanying this paper represent the elements of the parasite partially or totally comprised within the leucocytes. In reality, most observers who have denied extension by way of the lymphatics have reached this conclusion *a priori* on account of the usual absence of adenitis in the neighborhood of the foci of actinomycosis.

Israël also admits extension by the arterial circulation, but not for the liver in the case he was describing, where he supposes that the fungus reached the spleen by way of the arteries and from the spleen to the liver by the veins. Koranyi believes that infection by the lymphatics is very rare if it even exists.

Therefore, from what has been said one may admit that there are four ways of extension of actinomycosis to the liver, namely:



(1) By continuity, in this case the focus in the liver is secondary to either (a) an infection of the digestive tract or (b) a pulmonary focus; (2) by the veins; (3) by the arteries, and (4) by the lymphatics. I shall follow this classification.

I have collected eight cases in which infection of the liver took place by continuity. In two the infection came from the appendix, two from the transverse colon, one from the ascending colon and duodenum, one from the right colic angle, one from the stomach and in the last case it was difficult to say whether the extension came from the lung to the liver and then from the liver to the kidney, or on the contrary from the kidney to the liver and then the lung. As the affection began with pain and a tumor in the right iliac fossa and as no mention is made of the lungs, the latter way of extension appears to me the most probable, regardless of the relative rarity of primary renal localization of actinomycosis.

I have collected eleven cases in which, according to my way of thinking, extension took place by the veins. These are cases in which there were no adhesions with the neighboring viscera, consequently extension by continuity is out of the question, and where neither the lymphatics or arteries could be incriminated because the lymph-nodes were not enlarged, nor was there the peculiar progress observed in invasion by the arterial circulation.

In none of the literature I have been able to consult has there been recorded cases of actinomycosis of the liver from extension from the lung. Such cases however do exist and Boari mentions two, those of Kauthack and Snow. Neither have I found reported cases of lymphatic extension, but, contrary to most writers on the subject, I believe this way of extension possible. For that matter, this point of view is well demonstrated by the writings of Pawlow-sky and Maksontow to which I have already referred.

With Israël, I believe that in the pyemic form of the process, the fungus passes first into the lymphatic vessels and then into the blood current and is carried throughout the organism. In the cases in which I admit venous extension the primary focus was invariably in the large intestine. In two it was perityphlitis, once appendicitis and once paratyphlitis. Twice the rectum was the primary seat of actinomycosis, once the ascending and once the transverse

colon and one the intestine in general with tumefaction of the mesenteric lymph-nodes.

There were four instances of the pyemic form of the process.

As to the cases of primary actinomycosis in the liver the case reported by Boari does not seem to me positive. This observer says: "I am led to believe that the parasite, which gained access to the organism by way of the mouth, then emigrated to the liver by passing into the circulation. This hypothesis acquires value, if it be recalled that the mouth is the ordinary entrance for the parasite." I do not very well see by what route the fungus could have followed to reach the blood-vessels from the mouth. Boari goes on to say that "the abscesses in the lung did not contain grains of actinomyces, they were metastatic abscesses due to vulgar pyogenic bacteria. Therefore, there was not extension from the lung to the liver."

In my opinion this reasoning is insufficient. The pulmonary abscesses which did not contain the actinomyces at autopsy may have contained the fungus before, and the latter, being less resistant than the bacteria present, may have disappeared from destruction due to the cells of the infected tissues or by the greater rapidity in the development of the other bacteria. Consequently, it cannot be affirmed that the hepatic lesion was a primary one and may very well have been secondary to the lung focus.

In Bristowe's case, the first symptoms do not seem to have indicated an hepatic actinomycosis. However, at autopsy no other lesions than those of the liver could be found so that it may be regarded as an instance of primary actinomycosis of the liver. Langhan's case seems to have been secondary to a focus in the stomach. The stomach had been weak since childhood and was invaded by the parasite which from this organ invaded the liver by way of the gastro-hepatic omentum as was made evident by lesions existing in the omentum and strong adhesions binding the greater curvature to the liver. In Moser's case it is probable that the primary localization of the fungus was in the bronchial tubes and lung, but it is difficult to decide whether the extension took place by continuity through the diaphragm or by way of the blood.

In Taylor's case, the antecedent intestinal symptoms and the adhesions of the liver to the colon, found at autopsy, show that the liver lesions were secondary to a process in the large intestine. This

is probably the same in Van der Straeten's case in which similar lesions were found.

Of the thirty cases I have collected the sex is given in twenty-eight, namely, eighteen men and ten women. The age of the patients varied between eleven and sixty years, but the majority were between twenty-two and fifty. There was a child eleven and a youth of eighteen, one male of fifty-three and two sixty years of age.

There was only one among these patients who was a farmer; the remaining twenty-nine had trades that did not expose them to infection from the actinomyces, and Taylor's patient developed the disease in London where he lived.

*Symptomatology and Clinical Types of the Affection.*—The symptoms of actinomycosis of the liver are not very characteristic and vary greatly from one case to another. The local symptoms are not always very marked, especially at the onset. Quite frequently the hepatic process seems to have been overlooked in the midst of concomitant phenomena and was only discovered at autopsy. Sometimes the gastric or intestinal symptoms predominate over those of the liver.

In other instances the affection assumes the form of pyemia, either early in the process or after a more or less long acute phase. I therefore will classify the process under three heads, as follows:

- (1) A form in which the dominating symptoms occupy the hepatic region = *the hepatic form*;
- (2) A form in which the dominating symptoms are related to the stomach or intestine = *the gastric or intestinal form*;
- (3) *The pyemic form.*

*The Hepatic Form.*—Sometimes one will be dealing with an abscess of the liver from the onset. In Hoeffner's case there was an abscess in the right hypochondrium; in Ullmann's case, the abscess extended from the costal edge to the umbilicus. In other cases the abscess develops later. In the case reported by Lüning and Hanan, the tumor appeared in the right side of the abdomen, and two months later an abscess developed. In Schartan's case the abscess was seated under the edge of the left costal margin, and at autopsy two liver abscesses were found, one in the left lobe which communicated with a cutaneous pus collection, and another abscess in the right lobe in the midst of the parenchyma.

The abscess in the liver may develop suddenly with pyrexia, as in Boari's case or be preceded by more obscure symptoms. In Eve's case there were tumor and pain in the hepatic region and a marked increase in the size of the liver which came below the ribs. The tumor was mistaken for a gumma. A fortnight later the tumor became painful, softened and was incised. Langhan's case presented pain in the epigastric region, a mild jaundice—the only case where this symptom was noted—and a large painless liver. Until operation was done the diagnosis wavered between an hepatic abscess and an echinococcus cyst.

In Taylor's patient the onset began with a tumor in the right groin, and a fortnight later another tumor appeared a little above; the pain varied in character; there was constipation but the appetite was good. A large tumor filled the right flank and the percussion dullness over it continued with that of the liver. In Van der Straeten's case the onset was also obscure, dull pain in the right side, change in the general health, evening temperature 102.2°F, morning 100.5°F. The lower part of the thorax projected, especially at one spot which soon became painful. A puncture made at this spot revealed the true nature of the process.

Three of these patients suffered from constipation; another had had a so-called perityphlitis one year previously, and another gave the history of a previous appendicitis (not operated on). Taylor's patient had suffered from indigestion before the hepatic process arose; he had been constipated for six months and had an attack of colic shortly before the liver symptoms developed.

*Gastric and Intestinal Form.*—I have notes of two cases in which the symptoms were at first gastric, namely, Langhan's case already referred to and Bristowe's case. This was a young woman who complained of the stomach for six weeks before coming to the hospital. She felt as if her stomach was always full. Three weeks after she had been in hospital she complained of an acute pain in the left side and this was followed by pleuresy and peritonitis. The affection of the liver was only found at autopsy.

Heller-Borgum's patient was a man who had had actinomycosis for three months; there had been an extensive dysenteric process of the large intestine, three abscesses developed in the liver and the

patient died of purulent peritonitis from rupture of the abscesses into the peritoneal cavity.

In Friedrich's case, the patient's illness began with vomiting, violent pain and inflammatory pelvic phenomena, anorexia and constipation. Then tumors of the abdominal parietes developed but did not communicate with the abdominal viscera. These were removed at two surgical interferences, the second resulting in a wound of the gut followed by a fecal fistula. The patient rapidly declined with chills, pyrexia and violent pain in the right hypochondrium. Cardiac dullness was increased. Death ten days after the first chill.

I include in the gastro-intestinal form the cases in which the dominating symptoms were seated in the region of the cæcum and appendix, and in all of them in which abscess of the liver occurred the diagnosis of the hepatic process was only made at autopsy.

*Pyemic Form.*—This form does not develop at the onset of the process. In Hebb's case the patient suffered for a month with pain in the limbs, diarrhœa, vomiting and pyrexia. When he entered the hospital there were signs of pulmonary induration and a pleural collection which was twice aspirated. Soon afterward symptoms of pyemia developed and death took place.

In Israël's case, when the patient entered the hospital, he had been ill for seven months with pain in the limbs with almost daily paroxysms of pyrexia beginning at noon and subsiding in the evening with profuse sweating lasting all night. The history showed that the patient had received a thoracic traumatism and later a tumor developed which went on to suppuration. After this, a large number of abscesses developed in different parts of the body. Israël says that blood examination was negative. The temperature was irregular. The first chill occurred six days after the patient was admitted and a second one occurred five days later. Death twenty days after entering hospital.

Boari's patient died with all the symptoms of pyemia.

The classification I have attempted is not by any means fixed. As will be seen some cases presented one form at the onset of the process and another form at the end of the disease. Other cases could not be readily included in this form.

*Complications.*—I here append the principal complications that have been noted: Appendicitis, three times; perityphlitis and para-

typhlitis, each once; peritonitis, five times; pleuresy, five times; abscess of the lung, five times; pyothorax and pneumonia, each once; pulmonary tuberculosis, once; involvement of the spine by the actinomyces, once; two stercoral fistulæ; actinomycosis of kidneys, four times; spleen, twice; uterus and adnexa, twice; and lastly, there was a psoas abscess in one and pericardial collection in another.

*Diagnosis.*—The diagnosis of actinomycosis of the liver is not easy. It can be suspected when a patient with a large liver is neither syphilitic nor ill with hypertrophic cirrhosis of the liver and has not lived in tropical countries. It is only when an abscess develops that a diagnosis can be made after aspiration of the pus. When the symptoms seem to be due to the stomach or intestine, the diagnosis will be still more obscure. However, if the symptoms do not seem to easily apply to an evident affection of the digestive tract, intestinal actinomycosis should be thought of and the liver carefully explored in order to detect any possible complication arising in this organ.

It is to be remarked that a certain number of patients suffered previously with affections the nature of which could not be made out from the history. If the case is one of pyemia when first seen it will be impossible to make a diagnosis of actinomycosis, and the possibility of infection by this fungus can only be suspected from the patient's antecedents.

Whatever may be the form assumed in a given case, aspiration of the hepatic abscess and examination of the pus is the only means we have of making a diagnosis and this will only be certain when the yellow grains are found, but if they are absent this is no reason for rejecting the diagnosis of actinomycosis.

*Treatment.*—Medical treatment, including massive doses of KI, is fruitless. The general health should be attended to and tonics, including arsenic, should be exhibited and KI may be given as an adjuvant. Constipation when present is to be controlled.

As soon as an abscess has formed in the liver, treatment is strictly surgical. Incision of the abscess will at least give a momentary relief to the patient. But a permanent cure must not be expected since all the cases so far reported have died. Rupture of the abscess into the peritoneal cavity or the development of pyemia naturally terminates in death at short notice.

## ACTINOMYCOSIS OF THE CÆCUM AND APPENDIX

By MANUEL HINGLAIS, M.D.

Formerly Resident Physician to the Hospitals of Lyons and of Bône, France

THE most constant starting-point—one might say the only one—is from intestinal actinomycosis in the ileo-cæcal segment which offers an excellent medium for the growth of the fungus. In Chiari's case, the patient did not present, during life, any morbid manifestation which could have led to the suspicion of actinomycosis. At autopsy, a round, grayish patch, one centimetre in diameter and five millimetres thick was found in the mucosa of the cæcum and was formed by a pure young growth of actinomyces whose filaments had penetrated and filled the subjacent Lieberkühn's glands. This is the most simple cæcal lesion of actinomycosis yet observed, the fungus still being on the surface.

Later on, when the parasite begins its onward march, it produces ulcerations of variable size which may attain the size of a silver ten-cent piece, although they average about the size of a lentil. These ulcers repose upon the mucosa and muscular layer of the intestine; their surface is lesser in extent than their base, hence the edges are undermined. At length the parasite by its progress perforates the intestinal walls. It is clear that if the evolution of the process were very rapid so that the organism would not have time enough to give rise to defensive peritoneal reaction, putrid peritonitis would be the inevitable result, but Choux explains the absence of peritoneal phenomena at this phase of the process by an eversion of the mucosa outwardly, thus acting as a plug.

This, however, is not the case. At the very onset of the evolution, from the time the fungus has perforated the mucosa, a peritoneal reaction starts in having as a result the development of dense adhesions which thicken the intestinal walls and bind them to the adjacent structures, in such a fashion that the parasite is offered a thick, resistant barrier that it must first destroy before it can reach other viscera in the neighborhood.

This defensive phase corresponds to the symptomatic phase of

the formation of the tumor and softening. On the other hand, still another form of defense takes place. In point of fact, it has been remarked that the tissues invaded and then abandoned by the fungus, sometimes even before it has completely disappeared from them, have a marked tendency toward cicatrization. For this reason the mycelium has been found in the thickness of the intestinal wall while a cicatrix of the mucosa indicated the point of entrance of the parasite.

All the sinuses are filled with cicatricial tissue at their deep end, hence the presence of hard cordlike tracts, made up of neo-formed tissue, which connect old and recent foci and are, in reality, the traces left of the road followed by the parasite in its progress.

In this progressive onward progress it is to be remarked that no tissue is respected; no matter what kind of barrier is opposed to the progression of the actinomyces it in the end becomes infiltrated and broken through by the fungus. We consequently are in presence of a pathologic action quite different from that offered by other infectious processes or special processes, such as tuberculosis for example, in which the bacteria follow the anatomic planes, and do not break through the fasciæ until a long time has elapsed or open into the blood-vessels until the later phases of the process. Here, this takes place from the start, the infiltration advances without respecting any structure whatsoever.

However, I would remark that the connective-tissue process arising in intestinal actinomycosis tends to occlude the vessels rather than open into them, and later on, when sinuses have developed, the vessels may serve as a road for the evacuation of the products of the actinomyces, as Kosenski observed in one instance.

This progressive involvement of the tissues, special to actinomycosis is due to the fact that the fungus introduces itself into the cells from without inward, then perforating their walls from within outward, the filaments of the fungus place themselves in contact with other cells that they in turn penetrate.

The phase of tumor formation is a phase of connective-tissue neoplasia. According to Partsch, the connective tissue is fibrous and granular, in the midst of which the pathogenic agent becomes fixed and proliferates. The phase of softening is the phase of destruction resulting from a decrease of the tissue vitality. According to Choux,



the actinomycoma is an infectious tumor in the same sense as syphilitic and tuberculous tumors of those arising in glanders.

If a softened spot in one of these products be opened, little fluid will be found and what there is, is usually clear, serous and sticky. The walls of the cavity are thick and hard. When bacterial association exists the cavity is anfractuons; when the actinomyces is in a pure culture the cavity is lined with granulation tissue which, containing the parasite, becomes detached and assumes the yellow color regarded as belonging to this infection.

It is to be recalled that the periosteum and bone are very rarely invaded by actinomycosis and the lymphatic system is never involved. Metastases of the parasite rarely, if ever, take place by way of the lymphatics, and follow a venous route when they occur.

When the affection is of long duration a peculiar form of cachexia takes place—*actinomycotic cachexia*— and the subject finally dies. At autopsy the intestinal coils are agglutinated and adherent to the adjacent viscera and abdominal walls. The intestinal walls present amyloid degeneration, likewise other of the abdominal viscera. From this fact it will be understood that the intestine is immobilized by its adhesions; that its own tissues are choked by neoformed connective tissue; and lastly, that its blood supply is decreased and at the end of the affection will be almost completely cut off.

*Incubation Period.*—It is hardly possible to assign an exact duration to the period of incubation of cæcal actinomycosis. However, it may be supposed to be long, because one finds that patients very often present phenomena of reaction quite a long time after they have been exposed to infection. Thus in a case of actinomycosis of the hand, Müller found that the first symptoms developed only after two years following a splinter of wood entering the skin and remaining embedded. When examined microscopically the splinter was found covered by growths of the fungus.

Ljungdren believes that the foreign body bearing the mycelium is necessary for its development and plays the part of a culture medium. The chances will therefore be greater for the development of actinomycosis the longer the bits of cereals or other matter serving as a vector remain in the wound.

It is also difficult to appreciate the duration of incubation on account of the latency of the early symptoms in the majority of

cases in which the process was chronic with a slow evolution. The disease may then be overlooked until the subperitoneal cellular tissue has become invaded, or when an associated bacterial infection is present, the formation of pus collections.

In the opinion of all observers the most constant starting-point of the process is the ileo-cæcal region and appendix, this being due, as I have said, to the prolonged stagnation of the detritus of digestion in the cæcum, which allows the fungus to become fixed on the intestinal mucosa and there develop and become encysted, and then to continue its reproduction which terminates in general infection, and to the morbid phenomena encountered.

*Symptomatology.*—From the viewpoint of symptomatic manifestations I shall divide the evolution of the affection into five phases, viz., (1) *Prodromes*; (2) *initial symptoms*; (3) *tumor formation*; (4) *softening, ulceration and sinus formation*, and (5) *cicatrization*.

*Prodromal Phase.*—This phase—usually overlooked—makes itself evident by dull pain, with indefinite localization, usually attributed to a dyspeptic state, or the symptoms of peritonism.

*Initial Symptoms.*—This phase corresponds to that of catarrh and should be regarded as the real onset of the process. In rare instances it may be sudden and by constituting the entire affection causes death with extreme rapidity. Colic and profuse diarrhœa arise. The stools may be mucous or dysenteric with blood and accompanied by abdominal pain but without peritoneal reaction. However, in some instances like the case to be reported, attacks of appendicitis with abdominal distension and vomiting have been observed. The duration of these attacks has varied from two or three weeks to as much as three months.

The diarrhœa is stubborn, while its more or less lengthy duration appears to be connected with the localization in the mucosa or muscular layer of the intestine. However, it must not be assumed that diarrhœa is constantly observed, because we know that both it and other symptomatic phenomena may be completely wanting, the intestinal lesion being only discovered at autopsy.

On the other hand, the diarrhœa may be interrupted by periods of constipation, but this is rather infrequent. After a somewhat long lapse of time—depending upon the rapidity of the evolution of

the infiltration—the pain subsides, and the affection enters upon its third phase. Until now, rectal or vaginal examination and abdominal palpation will not have revealed any sign in particular other than perhaps some slight pain on pressure.

*Tumor Formation.*—It is especially at the time of the appearance of the peculiar phenomena of this phase that the surgeon's attention will be awakened. It is then, and then only, that if he will follow and interpret all the phases and evolution of the process, an almost certain diagnosis will be reached.

Now, in point of fact, will appear those symptoms that seem to belong to actinomycosis of the cæcum and appendix. They correspond to the pathological phase when the fungus—ready to break through the walls of the gut—makes an onward progression by infiltration of the adjacent tissues without regard for anatomical barriers.

At the onset of this phase palpation will give rise to the sensation of a hard, deep, diffuse tumor, which is still independent from the abdominal parietes. The growth must be, so to speak, taken by surprise at this time, because the infiltration will extend to the abdominal parietes which then will form part of the tumor mass. The infiltration extends extensively, sometimes at once involving the entire right half of the abdomen, and always in a very short time, from the linea alba to the iliac crest. It is not uncommon for it to extend to the left of the linea alba.

As I have said, the tumor thus formed is hard, but the hardness is special, namely, woody or bony; by touch one has the sensation of an extremely resistant plane. The mass can no longer be moved in totality; it is fixed both laterally and in the depths; the limits are diffuse and difficult to outline. Over this induration the abdominal parietes are no longer depressible; it seems as if the subjacent viscera are solidly fixed, forming a mass adherent everywhere over the osseous plane.

All these phenomena undergo their evolution without pain in cases of pure actinomycosis. Palpation is painless as long as softening has not occurred. Let me mention however, that in some instances dull spontaneous pain occurs due to the development of adhesions, but with no other symptom of peritonitis strictly speaking.

At this phase rectal or vaginal examination may give some information. Sometimes one may recognize the starting-point of the tumor,

make out its shape or seize it between the fingers in bimanual palpation, but it will invariably give the feel of a special kind of hardness.

The infiltration—I insist on this point—extends always to the surface of the body, and when once it has involved the abdominal walls it seems to extend more in surface than in depth.

The skin covering the mass remains for a time normal, and although fixed and adherent it offers no trace of inflammation. But soon the process continuing its progressive evolution passes to the next phase which is made evident by new symptoms.

*Phase of Softening, Ulceration and Sinus Formation.*—Before describing this phase of the process, I would say that the abdominal lesions will always be spontaneously evacuated through the abdominal walls if no complication has already caused the patient's death. It is in this phase that the spontaneous or provoked pains of the onset reappear.

The physical symptoms are as follows: The skin covering the tumor assumes a special color which is not that of common inflammation; it is a violet blue, blending off from the centre toward the periphery and may even take on a slate color. This color, considered as pathognomonic by many observers was present over the areas of ulceration in the case I shall report. At the time the change of color of the skin occurs, softening of the underlying structures takes place. Grill maintains that the softening takes place in the middle of the tumor, but in my case it arose on the left side of the abdomen, and this is what occurred in several other reported cases. The first sinus opened in the linea alba while the principal mass was located on the right side.

The process of softening goes on rapidly; in Bostrom's case in two days the tumor, which was the size of a foetal head, had become entirely fluctuating.

The softened walls give a distinct sensation of fluctuation although they contain little fluid when there is no pus, that is to say, when there is no bacterial association. If the pocket is not incised and opens spontaneously, ulceration quickly follows. In cases of pure actinomycosis little fluid escapes when the incision is made and this liquid is not pus strictly speaking. It is at first mucous, clear and sticky with absence of yellow grains; then, when the tumor is

pressed in order to empty it, the fluid expressed is thicker, viscous and grayish-white.

If the softened points are incised before spontaneous ulceration has taken place a pocket will be entered with thick, hard walls, containing little fluid, lined with pedunculated or sessile granulations which may reach the size of a millet seed. Some observers maintain that the pus of actinomycosis has a special odor. In the many cases I have seen in different regions of the body I have never been able to discover any particular odor of the pus.

Following spontaneous opening or incision of the tumor, numerous sinuses develop which may last for years. The walls and apertures of these tracts are lined with granulations similar to those found in the softened cavities. They give issue, intermittently or continually, to a clear, sticky fluid—purulent when bacterial association is present—which contains the yellow grains from time to time.

When the process is to end in death of the subject, the general health declines, metabolism does not take place properly, cachexia appears and it is often at this time that constipation arises. This results from mechanical conditions. The degenerated and agglutinated intestinal coils no longer force the intestinal contents onward; the phenomena of denutrition result from the deep changes in the intestinal mucosa and other tissues of the intestine so that absorption no longer take place.

*Phase of Cicatrization.*—When, on the contrary, the process tends toward a happy issue, the induration progressively retrogresses from the periphery to the centre, the abdomen slowly regains its softness until only one or two sinuses remain, which in turn spontaneously close later on. The sinuses leave deep, indelible cicatraces, violet in color or black from pigmentation.

Finally the following facts are important to remember. When a case of actinomycosis does not become secondarily injected by bacteria the temperature remains between 98.6° and 102°F. *The lymph-nodes are never enlarged, and there are never any ascites.*

One word as to the peculiar manner in which sinuses behave in actinomycosis. They are always numerous and often communicate with each other both superficially and in the depths of the structures by means of hard cords which may reach the size of a goose's quill. The parasite seems to progress like miners hollowing out gal-

leries or like moles who from time to time come to the surface in order to throw out the earth they have burrowed through. Consequently, the orifices of the sinuses are scattered hap-hazard along the route followed by the fungus.

I will now give the notes of a case of caecal actinomycosis that I had occasion to follow in Professor Gangolphe's service.

Male, *æt.* twenty-three years, mason. Father died at fifty-four from some gastric affection. Mother alive and well. No brothers or sisters. Patient has always been well. Three years ago, at 11 A. M. while at work he was suddenly seized with dull pain low down in the abdomen; the pain increased so that he was obliged to give up working at 5 P. M.

At this time the patient noticed that the hypogastric region was hard but he states that it was never so hard as at the present time. He had violent colic with false desire to go to stool. The colic lasted four days but at the end of a week he was able to return to work.

For the following fourteen or fifteen months he felt absolutely nothing in the abdomen which had become soft. Ten months ago when doing his military service he was seized with a second attack of violent pain, but this time the abdomen was "as hard as wood but not distended." The hardness was seated below the umbilicus. He was treated by laxatives and a milk diet. He was discharged well in three weeks, but the abdomen had not become soft as it did after the first attack and remained somewhat hard.

When he returned home in September he suffered from attacks of colic every two or three weeks which lasted one or two days at a time, and after each attack the abdominal induration increased and did not retrogress in the intervals of the attacks.

In March of the following year he had an attack lasting one week, in May another and then the induration progressed until he entered hospital on June 6.

Since the second attack the patient noticed that he was losing flesh and strength. He also stated that the induration was at first much more considerable in the right side, the left having become hard much later. He has also periods of constipation but no diarrhoea.

When admitted, the abdomen was found indurated from Pou-

part's ligament on the right to the same region on the left, and from the pubis extended up the linea alba to the extent of about ten centimetres. Its upper limits were oblique from below upward and from right to left—higher on the left side. An area of softening appeared above the pubis.

Professor Gangolphe made a diagnosis of phlegmon of the space of Retzius, because from the history of the case and the data obtained by rectal examination he suspected a former appendicitis which had resulted in a pus collection, and since two areas of softening had now developed, one on the external border of the right rectus and the second in the mid-line above the pubis, operation was decided on.

The first mentioned softened area was incised and opened into a pocket containing a little fluid. The cavity felt as if lined by granulation tissue. But as the subjacent structures seemed to be normal, the peritoneum was not opened. A second incision, above the pubis in the mid-line, was made and this gave issue to about a soup-spoonful of purulent viscous fluid but no yellow grains were seen in it. The deep perivesical fat seemed normal so the exploration was not carried any farther. Hoping to find the starting-point of the lesions, a third incision was made along the left border of the left rectus, but as all the underlying structures appeared normal all the incisions made were simply drained.

For a few days the patient's temperature ranged between 102° to 103°F. The urine contained albumin; there were some symptoms of cystitis. The general health improved.

From July 31, numerous sinuses developed successively as follows: One below the left femoral arch and then one above at about one finger's breadth apart, one in the left inguino-scrotal region, a second one quite near the latter, one near the root of the penis on the left side of the organ, and lastly, one near the umbilicus. At the point where these sinuses occurred the skin became a blue-violet color.

During all this time the abdominal induration extended and at the time of the second interference the right anterior superior iliac crest seemed to be involved in the process of infiltration.

On account of the latter lesion operation was done on December 15. At this time Professor Gangolphe suspected cæcal actinomycosis and eliminated the diagnosis of tuberculosis on account of the absence

of fungous material at the orifices of the sinuses. A deep suppurating neoplasm was also eliminated from the absence of symptoms of intestinal occlusion and bloody stools. The idea of actinomycosis was suggested by the peculiar combination of inflammatory phenomena with a neoplastic aspect and the development of sinuses near the drains. The sinuses were first explored and only one, that near the umbilicus, was found to be deep.

The operation was as follows. The tumefaction near the anterior superior iliac spine was incised and this gave issue to pus containing yellow grains. These were collected for examination. A second incision in the mid-line from the umbilicus to the pubis, including the prevesical fat. A third incision on the right along the border of the rectus. These three incisions extended into the subperitoneal fat. A fourth incision below the right femoral arch.

After curetting away the granulation tissue, all the incisions were united by drains, the first with the third and fourth, the second with the third (see Fig. 1).

Dressing moistened with a 1 per cent. KI solution, and irrigation of the wounds with the same solution. The latter was reduced in strength to 1:200 later on. Fowler's solution *per os*.

Histologic diagnosis made by Professor Dor, revealed the presence of typical actinomycetes.

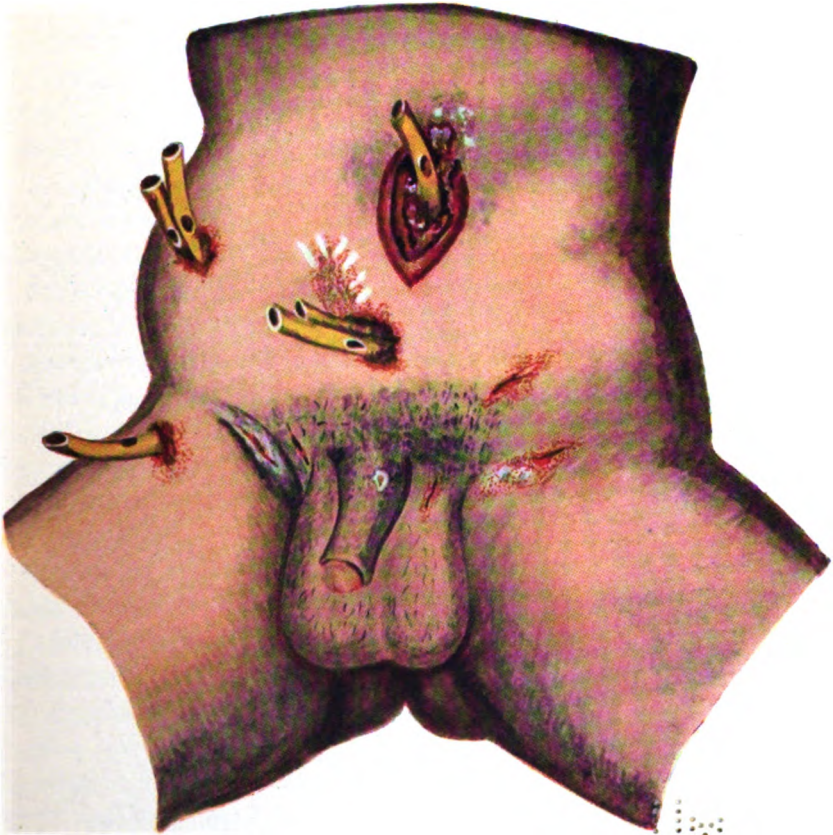
After the last interference the patient's health rapidly improved although another area of softening was incised in the right inguinal fold, the patient made a long convalescence with ultimate recovery.

*Complications.*—When giving the symptomatology, I endeavored to describe the perfectly regular progressive chronic progress of an actinomycosis devoid of any complication, but it must not be supposed that this is the usual evolution. Quite on the contrary, from the very onset or during the evolution, complications are prone to arise. Far from enlightening the diagnosis they may be the means of causing much perplexity.

The complications are first of all a tendency to deep extension into the retroperitoneal region by way of the mesentery to the posterior abdominal parietes. I give this progress as a complication although other observers do not regard it as such. My point is that it is this deep extension that is in most cases the cause of another



FIG. 1.



After curetting away the granulation tissue, all the incisions were united by drains, the first with the third and fourth, the second with the third.

100

extremely serious accident—one that is almost always fatal—namely, venous metastases to the liver, kidneys, lungs, brain, etc.

Hepatic metastases are the most frequent of all, obviously because of the large mass of blood arriving from the intestine to the liver by the portal system. It is also the most serious complication of cæcal actinomycosis as it is the cause of death from peritonitis due to intraperitoneal rupture of hepatic abscess. Undoubtedly the process progresses more rapidly in the liver than elsewhere so that the surrounding structures have no time to react and form protective adhesions.

Now, if the evolution of the secondary hepatic abscess is more rapid than other metastases, it has become complicated by bacterial association and therefore develops like an hepatic abscess having an infectious origin. Since the intestinal bacterial flora are legion in the intestine it is not to be wondered that secondary bacterial infection occurs. The result is the formation of pus, pyrexia, etc.

Perforation into the bladder of intra-abdominal collections due to actinomycosis of the cæcum and appendix have been reported. Another complication is extension of the lesions to the perineum with sinus formation around the anus and from here extending to the skeleton and even involving the hip-joints. Psoriasis is not an extremely rare complication of cæcal actinomycosis.

*Evolution and Duration.*—The evolution of actinomycosis in general, and that of the cæcum in particular, is essentially chronic. It usually is so from the onset, but it may occasionally assume an acute course. On the other hand and quite generally, the chronic evolution is interspersed with acute attacks. In other cases the affection proceeds by stages, especially at the onset, and in the intervals of these attacks there is an appearance of recovery, the patient seems to be completely restored to health, the tumor that was present disappears when, with the advent of some intercurrent affection, all the symptoms of actinomycosis return and the process continues its evolution. This happened in Guder's patient after an attack of influenza.

Another evolution is that simulating recurring attacks of appendicitis.

The duration of cæcal actinomycosis varies from one to about four years, rarely less than eighteen months to two years. Rapid evo-

lution of the process may be said to be exceptional, and as an absolutely unique instance I may mention Sokoloff's case where the patient died twenty-four hours after the onset of the symptoms.

Death usually occurs from progressive cachexia; otherwise the patient may die from purulent peritonitis usually due to metastatic abscess of the liver bursting into the peritoneal cavity.

*Diagnosis.*—With the data we possess, is it possible to make a diagnosis of actinomycosis of the cæcum and appendix? Can this be done at the onset of the process? At what phase of the disease may it be done with some degree of certainty?

To the first question I would reply that certainly the diagnosis can be made, because if all other data are wanting, a microscopical examination will clear up any doubts. I do not believe that the diagnosis can be made with certainty in either the first or second phases of the process. At this period of the evolution the symptoms are common to several intestinal affections, and even when one has a sufficiently certain anamnesis, a presumption may be had, but nothing more.

On the contrary, in the third phase, I believe that the evolution is typical enough for making a correct diagnosis. What should guide one is the tumor with its peculiar hardness, the progress of the infiltration toward the abdominal walls and its extension in surface without any distinct limits; the fixity; the special color over the areas of softening in the skin; the opening of the areas and the numerous sinuses resulting with their peculiar aspect; the small amount of fluid obtained when the areas break down or are incised; the great persistency of the sinuses; the slight pain; the presence of yellow grains; and lastly, the chronicity of the process and apyrexia.

*Differential Diagnosis.*—Let us now consider those affections with which a differential diagnosis must be made.

Acute or recurring appendicitis may be the cause of mistake during the entire duration of the first and second phases of actinomycosis, but they can be eliminated on account of the pain which is generally far less marked in actinomycosis and the development of the hard tumor in the latter affection.

Purulent cystitis due to emptying of the cæcal process into the bladder occurred in one case and gave rise to a mistake in diagnosis, but this we think could have been avoided had a rectal or vaginal

examination been made as the tumor or induration in the cæcal region could not have escaped attention. Likewise, the absence of pain should have attracted attention as pain is always considerable in bacterial infections of the bladder.

Tuberculous peritonitis offers several points of resemblance with actinomycosis of the cæcum. Like the latter process it is chronic, but the absence of ascites, the uniform dullness contrasting with the "chequer-board" dullness of tuberculosis of the peritoneum, the absence of plaques and isolated lumps typical of tuberculous infection, and the negative findings in the lungs should rule out tuberculosis. And into the bargain, sinuses are rare in tuberculous peritonitis.

Fibromata of the abdominal parietes cannot be compounded actinomycosis of the cæcum, although they are not painful, because they are, unlike actinomycosis, completely movable and they rarely ulcerate the integuments. They are more common in women during the sexual phase of life and often develop after labor. It is true that they often arise in the neighborhood of the femoral arch and on the external border of the recti, but they are distinct in outline and oval in shape. Their connection with the fascia and muscles is made evident by their fixity at the time of muscular contraction and their mobility when the muscle is relaxed, in which they are absolutely different from actinomycosis.

Sarcomata of the iliac bone might at the onset be a cause of mistake, but one will recall that in actinomycosis of the cæcum the neoplastic phase or rather the phase during which the tumor does not ulcerate, is of short duration and sinuses then develop, the contrary being true of sarcoma.

The diagnosis which, perhaps, presents the greatest difficulty is that of phlegmon of the abdominal parietes resulting from cancer of the intestine. In this case the intestinal disturbances—constipation, occlusion—usually, if not always, precede the development of a phlegmon in the infra-umbilical region. But when sinuses have developed, the nature of the morbid process will be made clear by the aspect presented by the granulation tissue lining them.

As to tuberculosis of the cæcum, besides giving rise to symptoms of recurring appendicitis, it may assume the form of a tumor in the right iliac fossa.

*Prognosis.*—Moosbrugger regards the prognosis of actinomycosis of the cæcum as very bad. Partsch is of the opinion that the prognosis is related to the evolution of the process; if this progresses rapidly to the surface the prognosis is relatively good, because it may result in spontaneous or surgical recovery. If, on the contrary, the process progresses in depth the result is apt to be fatal because surgical interference will be incomplete. The prognosis is bad when metastases have taken place. Grill maintains that bacterial association may be a happy event and that after its advent a spontaneous recovery may ensue. This tendency to recovery is due to the fact that the actinomyces have little resistance to bacteria and are killed off by them.

I have been unable to make statistics in respect to the prognosis because the writers I have been able to consult have used the same cases several times for establishing their statistics, but I think it safe to assume that the mortality at the best may be estimated at 80 per cent.

As to *treatment*, it is, of course, surgical. Operation should be resorted to early, and consists in as complete destruction as possible of the existing lesions. Medical treatment should be instituted at the same time, this consisting of the internal exhibition of KI, arsenic and calomel. General tonic treatment should not be overlooked.

## ACTINOMYCOSIS OF THE SKIN

By FRANÇOIS MONESTIÉ, M.D.

Formerly Interne of the Hospitals of Lille; Member of the Anatomic Clinical Society of Lille, etc., France

---

ACTINOMYCOSIS of the skin has no special etiology, but the manner of contagion is often peculiar. The seat of predilection is the face and to a lesser extent, the hands. In the case of the face the infection takes place by cereals or by meat. A carious tooth gives entrance to the fungus to the tissues, which reaches the skin and subcutaneous cellular tissue and there proliferates. An erosion of the buccal mucosa is also a point of entrance for the parasite, but in many cases the agent of contagion and the point of entrance remain unrecognized.

Infection of the skin of the hands takes place from cereals, wood, meat and cattle-skins which bear the fungus, and an abrasion opens the way to the fungus into the body. But the point of entrance is often unknown on account of the property possessed by the parasite of living for a long time in the tissues without giving rise to any reaction so that the patient will have forgotten the circumstances attending the contagion.

Adults are most frequently affected, the influence of sex, occupation, etc. appears to have little bearing on the process, although in my own experience I have had more cases in women than in males, but I attribute no importance to this fact. Other observers have met with the contrary. It is evident that farmers, drovers, butchers, hostlers and tanners are more exposed to contagion.

*Symptomatology.*—Cutaneous actinomycosis may be either *primary* or *secondary*. In many cases the secondary lesions of the skin have no importance, in others they may occupy the foremost place. Let us suppose, for example, that a patient presents a very limited focus of osteitis of the lower jaw due to actinomycosis; the lesion may extend to the skin which will become, so to speak, the essential part of the clinical history of the case. Whether primary or secondary, cutaneous actinomycosis, which has had time enough to undergo its evolution, will offer the same characters.

*Onset.*—The subject, after the receipt of an abrasion of the skin will notice a small, regular, rounded lump, sometimes situated in the subcutaneous fat. When the process is seated in the neck it may be overlooked. This little tumor is painless to pressure, but it may give rise to neuralgic pain in the teeth when the fungus has invaded the organism by a decayed tooth. The pain is not distinctly defined and may extend over the head or cheek.

When the onset is sudden the patient suspects that he has a dental abscess, especially if he has already suffered from one. But instead of retrogressing, the affection becomes progressively worse. By the end of one or two months, sometimes more, the tumor having insensibly increased in size raises up the skin, projecting more and more under the cutaneous surface, becomes adherent to the dermis which silently inflames, and the lesion slowly reaches the phase of full development at which time the patient usually seeks medical advice.

*Phases of Full Development.*—We have here to consider the local and general symptoms.

*Local Objective Symptoms.*—At this phase of the process the lesions present one of two aspects, namely, the *gummatous* and *anthracoid* forms. They can be distinguished from each other by the two following characters: In the gummatous form there are cavities recalling tuberculous or syphilitic gummata, containing quite a lot of pus and each presenting a few sinuses. The anthracoid form presents a large number of sinuses which give issue to very little pus. The lesion looks like a sieve. The gummatous form is the commonest, and I shall base my general description on it.

The integuments at first become pink, then red and lastly wine-color. The skin becomes thin and shiny, and then sinuses develop giving issue to pus in very small amount. Some of the sinuses close after a time, others persist and new ones form. Slowly the process progresses at the periphery; frequently an isolated sinus will open at a point quite distant from the primary focus because it is the depths, in the subcutaneous cellular or intermuscular tissue that the lesion extends, only to break out elsewhere to develop into a new centre of suppuration. By this mechanism it is possible for a vast cutaneous region—the cheek for example—to become the seat of very numerous sinuses.



The epidermis of the involved areas desquamates in five lamellæ especially at the borders of the lesion. Here and there are to be seen little black crusts formed by concrete pus and dried blood undermined at the edges, adherent at the centre and occluding the opening of a sinus. They become detached after a time or they can be easily removed, thus establishing the permeability of the sinus. When they are removed gelatiniform granulation tissue can be seen to project from the aperture of the sinus; they are semitransparent, translucid and soft. In one of my cases after KI treatment, this granulation tissue projected above the surface and formed a lesion like a comb of a cock. The little crusts referred to are often adherent to these granular elevations so that the general aspect of the lesion is mammillated. These numerous elevations are separated by furrows.

The furrows are of two kinds: Some are small and shallow and are the most numerous; others, on the contrary, are much more accentuated and separate larger lesions formed by the union of several small ones. The edges are almost always elevated and are quite distinctly separated from the healthy structures.

Such is the general aspect of the lesions of cutaneous actinomycosis, but there is yet another very important sign offered by the skin of the involved area. Our attention was drawn to it by Professor Derville who, I believe, was the first to describe it, and when it exists it can be regarded as pathognomonic. This sign consists of peculiar looking spots scattered here and there, offering the following characters: (1) They stand out against the rest of the lesion on account of their color. They are dark spots varying according to the more or less accentuated general color of the lesion. If the lesion is pale the spots are bluish-red or violet. If the general tint of the lesion is darker, the spots take on a slaty-black color. (2) Their size varies from that of a lentil to a pin's head, and they are regularly round although not always so. A white point may be seen at their centre looking like folliculitis. (3) At their level the epidermis appears to directly cover the granulations, and the translucid aspect that the lesion offers at this spot is due to the intimate relationship of the epidermis and soft fungous masses which form the mass of the actinomycosis.

These spots, in other words, appear to correspond with those points where the abscess pocket is the thinnest. What confirms this

opinion is that I have several times seen sinuses develop at these spots. The importance of these spots is particularly shown in the two following cases.

In the first case, after carefully collecting the pus for microscopical examination which was done by Professor Angier, I was astonished at the negative report made after long and careful examination. But the spots described were present with all their characters and the diagnosis of actinomycosis was maintained. Another examination of the pus finally revealed the actinomyces and the clinical diagnosis was confirmed.

The second case was negative but for this very reason I believe that it confirms the diagnostic value of the spots. The patient, a male, presented a lesion on the lower jaw that Professor Guermontez believed to be actinomycosis and referred the case to Professor Derville and myself.

The patient, *æt.* twenty-three years, was a bank clerk. Three months previously he had pain in the teeth and then a small lump appeared at the site of the pain, which progressively increased in size. At the end of a month a sinus developed, giving issue to blood and pus. One injection of carbolic acid solution was enough to close the opening of the sinus. A second sinus developed two months later giving exit to blood and pus. At the same time there was pain, the skin became red, but the spots referred to were absent. A negative diagnosis of actinomycosis was made. Several examinations of the pus proved negative and confirmed the clinical diagnosis.

These two cases show how useful the presence or absence of these spots may be in the diagnosis of actinomycosis of the skin.

*Palpation* of the lesions is also important. Two zones can be made out, a central superficial one, and a deep peripheral zone. The former is the projecting portion. It is soft, evenly elastic and smooth to the feel—in other words, it gives the sensation of very thin skin raised up by fungous masses. The sinuses develop in this central zone. Fluctuation may be detected at some spots and this may even extend from one border of the lesion to the other but it is never very distinct and always feels more like fungous material.

The second zone completely surrounds the first, limits it at its deep part and serves it as a base of implantation. It is very resistant, almost woody to the feel. It extends one or two centimetres

beyond the central zone and is from one half to one centimetre in thickness. It occasionally seems to adhere to the underlying structures but in three of my cases I was able to assure myself that it was confined to the subcutaneous cellular tissue after KI treatment had softened it a little. This indurated portion does not project above the surrounding skin and its color is less marked than in the central zone. The lesions are suppurative, but the pus is peculiar in type, with well-marked characters.

*Subjective Symptoms.*—There is about only one subjective symptom and that is *pain*, which may be spontaneous or provoked. When spontaneous it is intermittent, that is to say, it occurs in paroxysms. This pain occurs up to the time of sinus formation and ceases when the discharge begins. It recommences when another lesion begins to develop and continues until it breaks down in its turn.

Pain is provoked by pressure but usually it is rather mild because the tissues have a very limited vitality. For that matter, the lesion being chronic and riddled with sinuses there is no motive to provoke pain except when spontaneous pain exists whose intensity is greatly increased by pressure.

*General Symptoms.*—Less than that of the viscera, cutaneous actinomycosis is a debilitating affection. It is likewise probable that the fungus attacks people in a low state of health in many instances. In itself the process has a nefarious action on the organism, either directly or indirectly. It is clear that the actinomyces must borrow the necessary elements for its nutrition from the organism and above all it must deposit its nutritive detritus which is taken up by the blood, therefore exercising a nefarious action.

A more powerful cause of debility is the indirect action of the process. The patients suffer; they have days of repose followed by as many days of pain during which they do not sleep or eat. At the same time there is pyrexia. The tongue is coated and there is headache and little by little their strength declines. Consequently, these subjects progressively emaciate and reach a state of depression such that, in the more serious cases, death may result. The mental state is sometimes more involved than the physical on account of the long duration of the process.

*State of the Lymph-nodes.*—All observers state that the lymph-nodes are not involved in actinomycosis. I cannot subscribe to this

statement. Although I have no formal proof that the lymphatic system can be infected, the cases I have seen certainly permit me to doubt, because in all I was able to detect enlarged lymph-nodes. These were slightly tumefied, painless and few in number but their existence at parts of the body where their presence cannot be detected in a normal state, cannot be doubted.

In one case the indurated lymph-nodes were symmetrical, although the lesion was unilateral. In two others there was an enlarged regional lymph-node and after incision one disappeared. In still another case a single enlarged lymph-node was found on the opposite side from that of the lesion.

Adenitis is therefore not constant but can it be said that it does not exist or that it is not related to actinomycosis when it is present? Again, doubt is permitted and Ulmann has given the following explanation for the lymphatic lesions observed.

"Suppuration of the nodules of actinomycosis is not constant in man and offers nothing specific. In cultures made from the pus of actinomycosis I found the staphylococcus aureus and other organisms which undoubtedly developed at the same time as the actinomyces, or after it, in the tissues where it vegetated, the point of lessened resistance. Therefore, the enlarged lymph-nodes are not specifically enlarged because they do not contain the actinomyces, only ordinary bacteria."

*Evolution and Prognosis.*—The evolution of the process is essentially chronic; the lesions develop slowly and their onset is difficult to determine. In one of my cases, however, it was the very next day that a tumor of the lower jaw developed after the patient had passed the night in a barn. He was positive that nothing was wrong the day previously. In spite of this extremely rapid onset, the evolution of the process was nevertheless chronic.

The duration of the process is *illimited*, since there is little, if any, tendency to spontaneous recovery. If an appropriate treatment is not given for arresting the development of the actinomyces and bring about retrogression of the lesions, the process will continue until death occurs either from some intercurrent disease or a visceral complication or finally from progressive exhaustion, continued emaciation and final cachexia.

But a well-directed treatment will bring about a more or less rapid recovery, doubtful by surgical means but certain, I believe, by KI treatment which must be long.

The *prognosis* of the affection is at present good. The only stumbling-block being the diagnosis, but this can always be made if the symptomatology is well known. Cutaneous actinomycosis should be included in the class of curable affections.

*Diagnosis.*—The rarity of cutaneous actinomycosis and the absence of pathognomonic clinical signs have been the means of many mistakes. A large number of cases have been treated as either tuberculosis or syphilis. *When a lesion of the skin is met with that looks unlike anything that one has seen before, actinomycosis should at once be suspected.*

Taburet believes that a diagnosis cannot be made until the foci of actinomycosis have been explored by puncture which should reveal the presence of yellow grains in the liquid withdrawn. In other cases the microscope alone will settle all doubt. For myself, the diagnosis rests upon all the symptoms that have been described taken together: Dark redness of the skin, projection of the lesion above the surrounding integument, multiple nodules and numerous furrows and, regardless of what Rochet says, very distinct deep induration, numerous sinuses, etc. But there are two signs which I believe surpass all others.

The first one, mentioned by all observers, is the macroscopic and microscopic examinations of the pus, and it is a matter of importance to collect it properly. Therefore, I will briefly refer to the way this should be done. The pus should be collected in a narrow glass tube previously sterilized. The tube is placed over the aperture of a sinus. It is gently pressed along the track until a few drops of serous pus is seen to exude. The rarity of the pus and its serosanguinolent character should always lead one to suspect actinomycosis.

The pus covers the internal wall of the tube and then, if care be taken to wait for about twenty minutes, yellow, brilliant points will be seen standing out distinctly. They are about the size of a pin's head and are sometimes whitish-yellow. Microscopically, they will be recognized as actinomycetes.

The second sign is the one I have described which was so distinct in my cases. It seems to me almost more important clinically than

the pus because it is present before sinuses have developed—I refer to the dark spots first described by Professor Derville. Therefore, when a cutaneous lesion presents a wine color with slaty-colored points or sometimes bluish-red, scattered over the lesion, the size of a lentil, sometimes round, at other times elongated, semitransparent, giving the impression that there exists a gelatinous substance underneath the epidermis, and when the opening of a sinus later appears where the spots have been, an almost certain diagnosis of cutaneous actinomycosis can be made. I do not maintain that this sign is present in every case, but when it does exist it is very valuable for the diagnosis of actinomycosis of the skin.

*Differential Diagnosis.*—Suppurating tuberculous adenitis which causes redness of the skin and sinuses might be mistaken for actinomycosis. The adenitis seated in the lymph-nodes in actinomycosis never suppurates. The induration is less, and the woody hardness met with at the periphery of the nodules of actinomycosis is never met with in tuberculous lesions. In tuberculosis it may be said that each lymph-node has its own sinus while they are multiple in a nodule of actinomycosis. When no sinus is present fluctuation will be distinct in adenitis, a sensation of elastic resistance in actinomycosis.

In actinomycosis the characters of the pus are interesting. In tuberculous adenitis the pus is badly mixed containing white grumous flakes, sometimes very small, which might on superficial examination be mistaken for the grains of actinomycosis. But they usually differ by their larger size and color, which is grayish-white and not yellow. I would mention the case of a young girl who came under my observation. She had an ulceration in the submaxillary region and had been given an iodoform ointment. Yellow grains were found in the pus and microscopical examination had to be resorted to to settle the diagnosis.

The differential diagnosis with tuberculous gummata would seem to be easy and yet it has given rise to confusion. At the onset it may be difficult to make a certain diagnosis but cases are rarely seen at this phase of the process. When caseation has taken place the gumma is fluctuating, not indurated and the slaty-colored spots of Derville are absent. When the gumma has opened there is a cavity communicating with the exterior by a single orifice with thin, freely undermined edges and without gelatiniform granulations. It

is surrounded by a uniform violet zone, and in no way recalls actinomycosis. A gumma is perfectly indolent; there is pain in actinomycosis.

In its classic forms *lupus* could hardly be a cause of mistaken diagnosis, but certain atypical forms of the lesion may lead to confusion, such for example, as the rapidly developing type with sinuses. A common mistake is to take actinomycosis for a cold abscess, but a little attention on the part of the physician should avoid this mistake. When there are two zones, one with deep induration, the other soft and elastic, the process is actinomycosis; in cold abscess there is only fluctuation. The slaty-colored spots should be looked for.

*Syphilitic gummata* are all the more likely to be mistaken for actinomycosis because KI has a favorable therapeutic action on both processes. But a Wassermann if positive will settle all doubt. A gumma develops rapidly, it opens by a single circular opening with clean cut edges and a grayish or yellowish bottom. Cicatrization takes place quickly when treatment is instituted.

A differential diagnosis with cutaneous epithelioma will hardly be necessary, because the two lesions do not present much, if any, similarity. In epithelioma the regional lymph-nodes are often perfectly normal.

*Periosteal sarcoma* of the jaws may give rise to confusion when ulceration of the skin has taken place. But when it is recalled that the lesion is not in the skin, that the cutaneous process is due to compression of the neoplasm in the jaw with the result that the blood supply to the skin is interfered with—hence the ulcer—it cannot be mistaken for actinomycosis. Sarcoma reacts on the general health much quicker than actinomycosis.

Some observers think that a differential diagnosis with *rhinoscleroma* may have to be made. Such a mistake appears to me difficult to make. Rhinoscleroma is not common, it begins by the nose and later on invades the upper lip and adjacent regions, the nasal fossæ and larynx. It forms nodules of exceptional hardness but ulceration and sinuses are hardly ever met with. Finally rhinoscleroma is an aphlegmasic process, therefore confusion with actinomycosis is hardly possible.

*Treatment.*—This is surgical, medical and mixed. Incision of the lesions may be necessary but KI treatment is the only one to be

considered. It should be given in daily doses of two or more grammes and continued until recovery is complete. Three months are often necessary to obtain a result. I also use 25 per cent. KI ointment on the lesions.

By this treatment this is what I have obtained. During the first few days of treatment the patients complain of sharp pain; the tumors increase in size; the redness becomes more marked, and the slightest contact with the tumor causes pain. There is a sort of reaction similar to what takes place in tuberculosis when tuberculin is used, and this is one of the most curious points in the treatment of actinomycosis with KI. New sinuses form giving issue to a larger amount of pus, but it is less bloody and better mixed. Then the tumor mass collapses to a marked degree.

From time to time congestive attacks occur, each time followed by a notable improvement in the process. In all my cases these congestive attacks occurred during the first three or four days after beginning the KI treatment and the physician should be cognizant of this fact, otherwise treatment might be stopped.

If the pain should become very severe, the KI ointment may be replaced by one with bismuth salicylate until the process calms down. These congestive attacks rarely last longer than five or six days.

Soon the borders of the lesion lose their color; the tumor collapses more and more, and at length the sinuses close. Only a small and hardly apparent cicatrix remains.

One of my patients was pregnant but gestation continued to term, resulting in a living child. Darier and Gautier have also recorded a case of actinomycosis during pregnancy, treated by KI with a favorable outcome. Therefore, we may conclude actinomycosis has no untoward effect on the product of conception.

In closing I would say that injections of tincture of iodine have not given me good results. I believe that other observers have come to the same conclusions in actinomycosis of other viscera and regions. The X-ray treatment may be combined with the KI treatment.



## ACTINOMYCOSIS OF THE LACHRYMAL DUCTS

By GEORGE P. ROBERT, M.D.

Formerly Assistant at the Ophthalmological Clinic of the Faculty of Medicine of Lille, France

---

THE etiology of actinomycosis of the lachrymal ducts is most obscure and in almost all the reported cases no etiological data are mentioned. On account of the similarity of *actinomyces bovis* and that met with in the lachrymal ducts, it would seem that the propagation of the fungus must take place in the same way and in the same circumstances in man. It is not clear whether contagion can take place directly from animals to man but it must be exceptional.

Vegetable contagion of actinomycosis *bovis* is, on the contrary, not only probable but has been definitely demonstrated both clinically and experimentally. The transport by air of contaminated dust to the conjunctival surface and from here into the lachrymal duct has been considered by many observers as the most likely manner of infection. On the other hand, Gombert has shown that the parasite grows well in water; the fungus multiplies in sterile water, therefore it is possible that water may also be a vehicle for conveying the fungus to the conjunctiva. This hypothesis seems very probable when the relative frequency of this fungus on the conjunctiva of otherwise healthy subjects is taken into consideration.

In point of fact, it has not been generally admitted—or at least attention has not been called to the fact—that this parasite vegetates along with other ordinary bacteria on the conjunctiva, and Gombert's researches have shown that it develops one or two colonies out of eight or ten Petri's plates inoculated with conjunctival mucus. Consequently, this parasite may be found in the conjunctival cul-de-sac although actinomycosis may not develop in the lachrymal ducts.

The reason for this immunity cannot be found in an unfavorable influence of the tears since they constitute an excellent culture medium, according to Gombert. An attenuated virulence of the fungus from contact with the air may be admitted, because Duclaux has shown that this has an unfavorable action on the parasite and we know also that the contact of various bacteria found in symbiosis with the *actinomyces* has a most destructive influence on the

latter. The impossibility for the actinomyces to develop in cultures in which other bacteria of secondary infection are growing demonstrates this fact. These are circumstances which put the fungus in a state of lowered vitality—therefore lowered virulence—and explains the infrequent development of the parasite in the lachrymal ducts.

In order that their infection shall take place the fungus must, perhaps, find itself in more favorable cultural conditions resulting from a greater *primary* virulence of the parasite, due to a less prolonged contact with other bacteria or from a diminution of energy of the bacterial midst into which it has been transported, but it is certainly necessary that special conditions must be created for it in the ducts in which it will later undergo its evolution.

These conditions may be realized by a previous change taking place in the mucosa and Camuset long ago pointed out that concretions which form in subjects with chronic inflammation of the sac and duct or by a partial or total occlusion of the latter result in a stagnation of the tears. Consequently, in these circumstances, the parasite finding the necessary nutritive elements, a proper temperature for its culture and the exclusion of air, will develop in the form of spherical concretions just as it does on artificial culture media.

In Goldzieher's case an eye-lash was found in the centre of the concretion and had by its presence in the duct produced a more or less complete irritative occlusion which favored the development of the actinomyces.

In the case I shall report the patient had been submitted to catheterism of the lower right lachrymal duct and it was here that the actinomycosis, which was discovered three years later, developed. Catheterism after curettage of the duct and removal of the concretion demonstrated a manifest stricture of the nasal duct.

For that matter, the concretions develop at those points of the lachrymal duct that are physiologically narrow, these being at the external opening and the orifice of communication with the sac. The tumor formed by actinomycosis has, in most cases, been found at the latter point. In my case the parasitic mass appeared to have developed in the diverticulum formed at the entrance to the sac by the wall of the duct and the fold of mucosa known as Huschke's valve. The whole forms a kind of cæcum which is well arranged

for the development of the fungus. The lachrymal sac does not appear to be ever involved secondarily.

Actinomycosis has in 66 per cent. of the cases been met with in the lower lachrymal duct. Evetzky explains this predilection by the more active part played by the lower lachrymal duct in the elimination of the tears. For my part, I believe that this peculiarity can be explained by the fact that the parasitic mass is to be quite often found in the terminal portion of the duct, a portion common, so to speak, to both ducts since they open into the lachrymal sac by a common orifice situated in front of Huschke's valve.

The clinical signs of the occlusion only reveal themselves in the lower lachrymal duct on account of the declivous direction of this duct and the low situation of the infundibulum in which the tumor develops, so that the lower lachrymal duct is regarded as being the site of the process.

Actinomycosis of the lachrymal ducts appears to have a marked predilection for the female sex, as in almost all reported cases it has occurred in women. This fact is interesting, because in almost all other localizations of the fungus males have been decidedly in the majority.

*Symptomatology.*—Ordinarily, actinomycosis of the lachrymal ducts begins with slight lachrymation, although tenacious, which after a time is accompanied by hyperemia of the caruncle and the conjunctiva near the greater angle of the eye and edge of the eyelid in proximity to the involved duct. Lachrymation may be very slight indeed and this statement likewise applies to the catarrhal redness of the conjunctiva which may be confined to the caruncle or be completely absent. The lids are almost always glued together when the patient awakes in the morning, particularly at the internal angle. The patient experiences a continued tingling sensation, intensely disagreeable, as well as a feeling of constriction.

When the case is examined at the onset of the process only a slight eversion of the lid, due to a mild thickening produced along its free edge from dilatation of the duct, will be noted. By palpation over the lachrymal duct a small, sometimes painful, swelling will be felt; it is smooth, elastic and generally rather resistant. The lachrymal orifice is dilated and gives exit in most cases to a droplet of viscous liquid having a muco-purulent look. Rarely it may contain

the yellow grain of actinomycosis. The enlarged orifice of the lachrymal duct will sometimes allow one to perceive a yellowish mass in the duct, but usually the concretion can only be detected by exercising rather strong pressure over the latter, directed toward the orifice, and it will disappear from view as soon as pressure is relaxed. By everting the lid a slight tumefaction of that portion comprising the duct will be noted, giving a yellowish transparence to the part.

The parasitic mass continues to grow slowly and finally forms a small tumor which projects a little above the duct both in front and behind it. The skin rarely adheres to it but is redder over the tumor and the palpebral edge which becomes thickened and rounded. The lachrymal orifice dilates to almost twice its normal dimensions and gives issue to an intermittent drop of yellowish liquid. By palpation, the contours of the tumor can be fairly well made out, its dimensions varying from that of pea to a bean.

Then the inflammation becomes more extensive in the conjunctiva which may become totally involved. The subjective symptoms usually follow a parallel evolution, and the condition, which at first was tolerable, becomes a real infirmity.

When the duct has been incised the parasitic mass can be seen, usually seated in the lower portion of the duct whose dilated lumen forms a kind of diverticulum; this may acquire the aspect of a small pocket the size of the mass occupying its lumen and communicates with the lumen of the duct.

The parasitic mass is composed of agglutinated grains, being held together by a more or less abundant mucoid matter. The grains are usually yellow or green in color, but they are sometimes gray or even black. In Foerster's case the entire mass was black. The mass may enclose calcareous particles which become more numerous as time goes on and finally results in complete calcification.

Examination of the walls of the diverticulum shows that they are thickened, infiltrated, rough and usually lined by a thin layer of muco-purulent liquid.

*Case Report.*—Female, *æt.* sixty years, came to the clinic for lachrymation of the right eye that she herself thought was the result of a small tumor of the lower right lid. This tumor had slowly increased for eighteen months. The patient complained of a sen-

sation of pricking occurring more especially toward evening. The lids were rarely agglutinated in the morning.

On examination there was a slight hyperemia of the conjunctiva at the internal angle; the orifice of the lachrymal duct was everted and dilated to twice its normal size.

The tumor was about the size of a pea, projecting equally both in front and behind, rather resistant to pressure which did not flatten it. It was seated a little outside of the lachrymal sac on the course of and a little above the duct. Pressure over the tumor and duct gave exit to a droplet of cloudy fluid. Energetic pressure on the tumor did not make it visible at the lachrymal orifice. The skin did not adhere to the tumor nor was it red. The tarsal conjunctiva was somewhat hyperemic. Patient's general condition excellent.

Two years previously the patient had been treated at the clinic for lachrymation by a few *seances* of catheterism.

Treatment consisted of incision of the duct and curettage of the tumor which was found in a small infundibulum at the terminal portion of the duct near the lachrymal sac. By the curette the walls of the cavity were felt to be rough. Prolonged irrigation, cyanide of mercury dressings. Two weeks later recovery was complete and has remained so.

The parasitic mass removed was the size of a small pea, and was solely composed of agglutinated grains each the size of a pin's head and fifteen in number. Some were the color of iodoform but the majority were grayish, two or three absolutely black.

After suitable treatment and staining with picrocarmin or eosin—the details of which I need not enter—they were examined at + 500. The grains were seen to be composed by a central fibrillary mass stained violet. The filaments were interwoven in every direction, forming a delicate network. The filamentous ramuscles were distinctly arranged in rays and in several specimens were in contact with orange-stained rods. At the periphery numerous dichotomized filaments were seen with branches the same length as the main filament and slightly undulated. The focus was surrounded by a zone of conidia adherent to the radiated filaments. The conidia were separate from each other and I was unable to detect any at the terminal end of the filaments. The aspect of the preparations was exactly that of actinomycosis bovis.

Given this result, I divided the parasitic mass into two parts and made cultures of the fungus in a vacuum glass on potatoe, but without result.

I should add that I used the youngest grains for microscopic examination and all that were left were the brown or black grains, that is to say, the most degenerated. It is also to be remarked that the examination of a grain according to Gram's method showed the mycelium quite distinctly two days later, but not a conidium could be seen.

*Diagnosis.*—The diagnosis of actinomycosis of the lachrymal ducts can only be a certainty after macroscopic and microscopic examinations of the little tumor show the presence of grains containing the actinomyces. Some of the signs enumerated in the symptomatology—especially lachrymation—may be wanting, sometimes no purulent fluid can be expressed from the duct, even when considerable force is exercised.

At the onset of the process there may only be a trifling eversion and dilatation of the orifice of the duct. The tumor might be mistaken for a chalazion, but its site and the presence of other concomitant signs, such as hyperemia of the conjunctiva and a slight puriform discharge should lead one to suspect actinomycosis. Catheterization of the duct will relieve all doubt as it will reveal a more or less complete occlusion of the lumen of the duct.

A foreign body in the duct might very well give rise to the same symptoms but the patient's history and clinical evolution of the case should not leave any doubt as to the real nature of the trouble.

The presence of a mass in the duct outside of the sac, dilatation of the duct orifice and the fact that energetic pressure exposes a tumor at the orifice is enough to distinguish the process from lachryocystitis.

The prognosis of actinomycosis of the duct is essentially benign and all the reported cases ended in recovery after extraction of the small tumor mass. The cavity contracts, the lining mucous membrane becomes smooth, the secretion subsides as do all the other subjective and objective symptoms of the conjunctivitis. Recurrences are unknown. During the evolution of the tumor a small abscess may form with ulceration of the duct and spontaneous elimination of the mass of fungus. But even in these circumstances the termi-

nal portion of the duct and the sac remain intact. No case of lachryocystitis due to actinomycosis has ever been reported.

*Treatment.*—The treatment of this affection is of the simplest. After incision of the duct the parasitic mass is exposed and removed with the scoop. The walls of the resulting diverticulum are cleansed with gauze moistened with a 1 : 1000 bichloride solution and disinfection of the lachrymal tract is continued for a few days.

# ACTINOMYCOSIS OF THE NERVOUS SYSTEM

By EMILE JOB, M.D.

Military Medical School, Lyons, France

---

## PATHOLOGY

THE lesions met with at autopsy of subjects dying from actinomycosis of the nervous system involve both the cerebral tissue and meninges in most cases. However, for clearness of description I shall consider those of the brain and then those of the cord and its envelopes. As Poncet has pointed out, there is much similarity between tuberculosis and actinomycosis both from the viewpoint of pathology and symptomatology. There are two very distinct forms of lesions due to the actinomycetes, the first being distinctly localized in the form of one or several cerebral abscesses or a neoplastic formation; the second consists of diffuse lesions, as in the following case.

Male, *æt.* twenty-six years, gardener. The patient had actinomycosis of the right temporomaxillary region for which he had undergone seven surgical interferences and a prolonged KI treatment. The patient was sent to the country after the last operation and when he returned on October 24, the suppuration had practically subsided and the sinuses had in greater part closed. However, there was still considerable swelling of the right side of the face and head.

On November 4, the patient's condition became suddenly worse; from time to time he developed convulsive paroxysms with loss of consciousness, delirium and restfulness. The pupils were dilated, there was slight strabismus and it appeared as if the vision was greatly involved. The convulsions were frequent and the patient did not reply to questions.

November 6, the convulsive seizures were more frequent, occurring every two hours and lasting four to five minutes. During the intervals the patient was almost comatose. Very marked opisthotonos, pupils greatly dilated; œdema of papilla on right; patient lies on right side.

November 7, trepanation, which had been decided on, was given up on account of a sudden great improvement. The intelligence had



returned, the papillæ were normal, the convulsive seizures had disappeared, but the opisthotonos was still present. Four grammes KI.

November 8, opisthotonos more marked and prostration likewise.

November 9, temperature, 101.2°F. Improvement considerable; patient read the morning paper.

November 11, delirium with hallucinations, but patient is perfectly aware that his visions are illusions. Pupils normal, react to light; eyes more mobile than normally. Fibrillary movements of muscles of hands and some localized convulsions in some muscles of the forearm. Disordinated carphologic movements of all the limbs. Pulse rapid, regular, 110.

November 14, patient lies doubled up with contracture of extensor muscles. Retention of urine for past two days. Complains of violent pain in head and back. This condition remained the same until death on November 16, in convulsions.

*Autopsy, November 17.*—When the spinal canal was opened, pus was found between the dura and vertebræ, containing characteristic yellow grains. It seemed that this extra-dural purulent collection which existed at the lower dorsal region, was due to an accidental tear during the opening of the spinal canal which was very difficult. In point of fact, there was a vast intra-dural collection. The vertebræ were absolutely normal. The subdural space was occupied by a large pus collection from the occipital foramen to the sacrum. The pus was the same as that found outside the dura. The cord was soft and tore easily. The dura was very thick and adherent to the vertebræ and presented lesions of pachymeningitis.

The cranium was opened by removal of the posterior half and gave issue to a large amount of cloudy, reddish serous fluid containing numerous grains of actinomycosis. This liquid escaped from the cerebral ventricles, very probably on account of a tear made in the fourth ventricle, produced during the removal of the bone surrounding the bulb. The bones of the skull were perfectly normal. The cranial dura was normal over the convexity and in the temporal fossæ. Over the base there was pus infiltrated in the subarachnoid spaces from the optic chiasma in front, up to the origin of the cord behind. At this level the collection continued without interruption with the intra-dural purulent cavity of the cord. There was little fluid pus but concrete pus covered the dura and pia.

The pinna of the right ear was next removed at its base and the regions of the masseter and temple were dissected. No lesion in evolution was discovered, only a healed cicatricial lesion existed. The temporal muscle had disappeared being replaced by hard sclerous tissue. The cicatrices situated on the lower jaw were adherent to the bone, which itself was normal with, perhaps, the exception of one small area which presented a mild degree of softening, the nature of which was difficult to appreciate. It is, however, probable that the softening was not mycotic in kind. The masseter muscle showed traces of fibrous tissue.

*Histological Examination* (Professor Dor).—Sections of the cord revealed the presence of the mycelium of actinomycosis. Microscopically, there were numerous and varied lesions, but given the difficulty of appreciating how much was really due to lesions existing during life and how much was due to post-mortem change in a cord bathed in pus for twenty-four hours, it would seem premature to describe a myelitis due to actinomycosis.

I would merely say that the pia was thick, infiltrated by a considerable mass of leucocytes and that all the white matter in immediate contact with it was in a state of advanced disintegration. The blood-vessels were remarkably well preserved, and this fact permitted the distinct differentiation of actinomycotic meningitis from tuberculous meningitis. The canal of the ependyma and the gray matter surrounding it were also intact which was to be expected, since there was no pus in the fourth ventricle.

When one considers the size of the yellow grains, it may be questioned whether a grain may not have acted as a plug in the canal, thus preventing extension of the infection. The entire pia was surrounded by a thick layer of pus that was everywhere limited by the dura, being itself the seat of very distinct pachymeningitis, although less so than in cases of Pott's disease. The spinal nerve roots presented evident lesions.

It was in the gray matter between the anterior and posterior horns and at a certain distance from the canal of the ependyma that small foci of disintegration were found that we hardly dare to regard as pathologic, because we could not detect the fungus in them. The fact that the pus contained staphylococci makes these lesions less interesting.

Actinomycosis may occupy the depth or the superficial parts of the brain. In the former case meninges are adherent to the brain. The convolutions around the lesion are sometimes normal, at others flattened and cedematous. The brain does not usually offer signs of sharp reaction and congestion only exists around the foci. Bollinger is about the only observer who mentions hydroncephaly.

The seat of the brain lesion is variable. Bollinger found a tumor in the third ventricle, probably arising from the choroid plexus. Kelley, Orloff and Martin report cases of abscess in the white or gray matter near the rolandic area. Ponfick, König and Moosbrugger have found lesions in the temporal and occipital lobes and in the cerebellum. The fungus seems to attack indifferently either hemisphere and has never been found in the cerebellum as a single lesion.

Once established in the cerebral tissue the actinomyces provoke one or two very different lesions. In a single case—reported by Bollinger—the lesion did not assume the form of an abscess. These abscesses vary in size, some resembling simple miliary granulations, others reaching the size of a chestnut or even an apple. Their contents are composed of pus having a special odor, at times green, at others grayish, and may even assume a gelatinous aspect. In the latter circumstance it resembles rather a degenerated tissue than a true abscess and seems to be an intermediary phase between a neoplasm and pus. Ponfick gives special attention to the fungus found in the walls of the cavity. The fungosities and adjacent tissue are filled with small round cells and actinomyces. Further on there is a connective-tissue proliferation which walls off the process. This is the same type of tissue reaction that occurs in tuberculosis.

There is a great resemblance between neoformations due to Koch's bacillus and those due to the actinomyces, with the important difference that in the later process the blood-vessels are in tact.

In other ways actinomycosis resembles tuberculosis. Abscesses of all sizes develop by the same process that presides over the development of tuberculous abscesses. When several miliary neoformations unite, an abscess is formed and will enlarge very quickly if the fibrous capsule, of which I have spoken, does not develop and wall them off, although this may be only temporary.

Why does the pus of these abscesses vary in color from one case to another? In the first place, these abscesses are the result of metastases and it is known that the actinomyces is not an organism giving rise to suppuration, it is devoid of pyogenic properties, so that the diversity of characters of the pus should be attributed to the diversity of the pathogenic bacteria associated with the fungus.

Nevertheless, all cases of actinomycosis of the brain do not assume the form of abscess formation. The fungus may produce a neoplasia which macroscopically looks much like a myxoma or lipoma. When incised the growth gives issue to a gelatinous substance resembling the fluid of ovarian adeno-cystomata. In this gelatinous content, Bollinger found albumin and much mucine. Lymphoid cells in all degrees of disintegration were seen, and what is most important, numerous yellow grains. The walls of the cyst in Bollinger's case were composed of fully developed fibrous tissue and this observer does not hesitate to give this pathologic product the name of actinomycosis. We are quite in accord with the Munich pathologist.

In the ox, the normal type of actinomycosis is the neoplastic form. The sarcomatous variety has been met with in man and is in reality the result of tissue resistance; and since each tissue reacts in its own way the forms will naturally vary according to the localization of the process. Therefore the sarcomatous forms of actinomycosis, such as Bollinger's case, are to be distinctly separated from the suppurative forms.

With Rochet, I divide actinomycosis of the nervous system into two forms, namely, a localized form as met with in the brain and a diffuse form. The former may itself be divided into two principal types, *viz.*, that of abscess—by far the most numerous—and neoplasm. The diffuse form is especially met with in the cord, bulb and protuberance, but the rapid post-mortem changes taking place in these structures and the relatively few cases that have been observed prevent me from offering a detailed description.

The meninges are involved almost as often as the nervous tissue itself, although there are exceptions to the rule. They are not merely involved by continuity, but at distance as well. Nevertheless, the manner of the invasion of the meninges is of great importance from the viewpoint of the onset and ulterior evolution of the lesions. The

cerebro-medullary axis has three envelopes: The protective dura, the arachnoid which gives mobility, and the pia through which the structures are nourished. The arachnoid and pia are almost always involved together in inflammatory processes and like all other serous membranes they offer an excellent culture medium for pathologic bacteria and are, therefore, more frequently the seat of acute diffuse processes than the dura. The latter membrane, composed of fibrous tissue, reacts more slowly and surely, and by thickening becomes a barrier to parasites coming from without. Therefore, according to the manner of invasion of the actinomyces there will be either diffuse lesions or circumscribed morbid processes.

The case above reported represents the type of diffuse lesions, while a case reported by Moosbrugger represents a transitional case. Only a portion of the base of the brain was involved and the evolution of the process was much slower. Pachymeningitis had time to develop but there was nevertheless a puriform infiltration of the pia occupying a large portion of the base of the skull. The circumscribed lesion observed on the dura was a lesion of pachymeningitis. In Ponfick's case it was external, while in other instances it was internal. The thickening is primarily a reactional defensive process but little by little the fibrous tissue, like the wall of an abscess, becomes invaded by the fungus and then becomes covered by a purulent deposit.

I have nowhere found a special description of the arachnoid. This double layer of membrane is in contact with the pia and the dura. If the process is a slow one it soon becomes involved by the dural neoformations. When the lesion of the brain is a primary one, there may only be a reactional process in the pia, hyperemia at first and then the causative factor persisting, the lesion extends to the other meninges. On the other hand, in König's case the pia was infiltrated with fifteen to twenty small abscesses the size of millet seeds. But, unfortunately the case was one of secondary actinomycosis so that we cannot know what part may have been played by the fungus in the pathogenesis of the abscesses.

Before finishing with the meningeal lesions a word must be said about the ventricles and canal of the ependyma, these cavities being in communication by the cerebro-spinal fluid with the subarachnoid space and the free space existing between the arachnoid and dura in

the spinal canal. Hydrencephaly has been met with in at least two cases, and pus has been found on the floor of the fourth ventricle in several instances.

The vascular lesions require mention, especially those met with in the venous sinuses, which play such an important part in the cerebral circulation. Nothing can be said of arterial lesions as no mention is made of them by any observers in cases of actinomycosis of the nervous centres. Nevertheless, cedema and flattening of the cerebral convolutions have been noted, phenomena no doubt related to the state of the arteries or capillaries. They are probably the same processes present in cerebral tumors. They are merely disturbances of pressure without lesions of the vascular tunics. We are all the more authorized to put forward this proposition because in microscopical examinations no vascular lesions have ever been detected, even in foci where the nervous tissue was in complete disintegration. The rule that makes one of the differential characters of actinomycosis and tuberculosis reside in the integrity of the vessels in the former process is here again verified.

The study of the sinuses of the dura is otherwise interesting. I must particularly insist on one reported case, because the lesions met with, their continuity with identical lesions in the large vessels of the neck, are of great interest in elucidating the problem of pathogenesis that I am about to consider. It was in one of Ponfick's cases where, besides a thrombosis of the internal jugular, there was also a thrombus in the left transversal sinus. This sinus was invaded by a gelatiniform mass which had extended to the veins and had entered their lumen. The cavernous sinus was filled with pus.

Other observers have mentioned similar lesions and later I shall point out their importance. Unquestionably, the bacteria associated with the actinomyces play a part in these thromboses but for all that the fungus is found vegetating in the midst of the clots and thus uses an open road for reaching other organs. This is, for that matter, a lesion of continuity, because the actinomyces is met with everywhere along the track from the point where it began to vegetate up to the first focus in the brain. However, it cannot be otherwise because it is impossible for a grain of actinomycosis thrown into the circulation by the left heart to travel through the capillaries

and become fixed in the sinus of the dura. In all cases in which there have been lesions of the sinuses these were the result of an ascending thrombus in the veins of the neck, or else the actinomycosis had involved a part of the face or skull whose circulation is so intimately connected with that of the brain.

I have been unable to find any good description of lesions of the nervous fasciculi. This is to be regretted because certain clinical manifestations might be explained more clearly if nerve lesions had been more carefully noted.

#### ETIOLOGY AND PATHOGENESIS

Is there a primary actinomycosis of the nervous centres? Opinions differ, most observers deny that this can occur. The actinomyces is a pathogenic agent coming from without and therefore must have some means of entrance and in the embarrassing cases of cerebral actinomycosis—called primary for want of a better explanation—there are not sufficient reasons for regarding the focus as being really a primary lesion. The only real primary focus will have probably disappeared. But it has existed in some part of the body without having given rise to any symptom that could have indicated the date of its evolution.

When the actinomyces penetrate into the body they must first undergo a phase in their evolution at the point of entrance. Moosbrugger found a superficial localization of the fungus in 60 per cent. of the cases, while in the remaining 40 per cent. no point of entrance could be discovered. Of the 421 cases of actinomycosis collected by Illich, in 29 the point of entrance of the fungus could not be found. In Bollinger's case of actinomycosis of the brain which was closely studied during life and post-mortem all trace of a primary focus elsewhere in the body was wanting. It is certain that the actinomyces entered by some other way than the head since the lesion was deeply seated, and also there had been no traumatism of the skull. On the other hand, nothing proves that some superficial lesion did not exist which offered an access to the fungus for reaching the brain.

This case may be regarded as a primary focus in the brain but it is the only authentic one with which I am familiar. In all of the other cases the brain focus was secondary to lesions elsewhere and it

only remains for me to show how the process was able to reach the nervous centres.

We know that the fungus does not follow the lymphatics for its propagation in the organism. This has been proved clinically, pathologically and experimentally. As a bacteriological proof it has been maintained that the fungus is too large to pass through the finer lymphatic networks. On the other hand one of the most important symptoms of actinomycosis is the *absence of adenitis*. If occasionally some lymph-node enlargement has been found either at operation or autopsy, the adenitis was due to bacterial association and the mycelium or the conidia of the fungus have never been found in any part of the lymphatic system. Distant adenitis has been found only because any lymph-nodes existing in the actinomycotic mass will have been destroyed.

Extension by the blood current is quite another proposition. It would seem *a priori* that the fungus does not resort to it frequently, since in the primary neoformation that it sets up, it respects the blood-vessels. This manner of extension does however take place since thromboses and emboli have been found in the vessels up to the right heart. In a case reported by Zemann the primary focus of actinomycosis was in the Fallopian tube and from here extended to the liver, lungs and brain, causing abscesses in all, but since search for the parasite was negative in all the lesions this case must be regarded with suspicion. However, this may be, since a pyemic form of actinomycosis exists, the fungus must spread by way of the blood.

If the parasite follows this route it may do so in two different ways, namely, by thrombus or embolus. The yellow grains have been met with in the internal carotid and actinomycotic abscesses have been found in the myocardium of the left heart. Now, if one of these abscesses should rupture into the ventricle, the entire organism and the nervous centres in particular will be exposed to contamination. On the other hand, in nearly all reported cases of brain lesions a pulmonary lesion was the starting point of the infection. It therefore seems probable that the fungus reaches the pulmonary veins at their origin and then is transported to the left heart and from here is thrown into the general circulation. I would



insist upon this frequency of coincidence of pulmonary actinomycosis with cerebral infection by the fungus, in cases of generalization.

Let us, in fact, suppose that the parasite is brought from some part of the body by the venous circulation, in order to reach the brain it must pass through the pulmonary circulation, but the lumen of the pulmonary capillaries is small, therefore the fungus becomes fixed in the lung and only secondarily penetrates into the arterial circulation.

A word as to the form in which the parasite is found in the blood. Let it be simply recalled that Marchand maintains that propagation takes place by interference of the cells, since he saw the parasite in the cells in the form of filaments longer than the cells themselves. Whatever may be thought of this theory, it is an established fact that there are actinomycotic emboli and that these emboli may localize in the brain, there producing lesions have a defined symptomatological evolution and pathology. Distant propagation by the veins must be less often observed. In fact, it would certainly seem that the veins of the face and neck which have such intimate connection with those of the brain can alone give rise to the phenomenon.

However, the veins offer traces of the passage of the parasite very often; its presence in them has frequently caused thrombosis and usually they were lesions of continuity. The actinomycotic process arises in the primary lesion; it invades a vein, a clot forms and serves as a vehicle for the fungus to reach the brain. Unquestionably this manner of propagation is not employed when the primary lesion is seated at a great distance from the brain. I have only found it in cases of actinomycosis of the head and neck.

Numerous are the routes, in fact, that the parasite may select for extending from the face or neck or even the apex of the lung up to the base of the skull. The skull and vertebral column form a barrier for protecting the nervous centres, but it is pierced by numerous foramina destined for blood-vessels and nerves to pass. The free space left by the nerves and vessels is filled with connective tissue. The nerves are surrounded over a considerable extent by a meningeal diverticulum and the optic nerve especially is covered up to its terminal part. But a more complete examination will reveal another route. I refer to destruction of the bone tissue by the fungus.

The connective tissue is one of the favorite medias of the fungus; it is by the connective tissue that the fungus penetrates the most frequently. Primarily actinomycosis of the viscera is rare, the perivisceral cellular tissue being the first structure involved. The actinomyces developes with the greatest ease in the connective-tissue spaces. They follow them and thus slowly invade the adjacent regions. The muscles disappear, the vessels open, become thrombosed and the process still continues. Thus it may reach the base of the skull. Here the connective tissue, although less abundant, nevertheless exists, filling up the spaces left by the vessels and nerves. The sheath of the optic nerve becomes thickened, fibrous, attempting to limit the abscess but soon the meninges and brain become in turn involved in the process.

It has been known for a long time that the bones can be invaded by the fungus of actinomycosis, the lesions being direct extension, and not a few competent observers maintain that there exists a primary actinomycosis of the bones. This question, however, is of little import since the result will be the same. I would first remark that bone actinomycosis is far from being as frequent as that of the soft structures. Bone forms a barrier to the extension of the process, but it is not always the case, and when the bone is involved superficial or deep lesions arise.

As soon as the periosteum is destroyed nothing prevents the onward progress of the parasite and the lesions assume large proportions. In this respect there is a curious fact to be mentioned, namely, that the periosteum does not react and thus allows the development of vast superficial lesions which later extend in depth. All these bone lesions have been met with in the regions which interest us. Langenbeck met with a case of mycotic caries extending from the last dorsal vertebra to the promontory. In another case the purulent focus extended from the atlas of the fourth dorsal vertebra. The basilar apophysis and the great wing of the sphenoid were eaten by foci of actinomycosis and in spots through their entire thickness.

The skull and spine are consequently no obstacle to the invasion of the viscera they contain. The actinomyces rarely attacks the upper jaw although Quenet was able to collect sixteen cases. Five of these patients died from invasion of the nervous centres by the fungus. These figures are enough to show the importance of lesions

of this bone in the pathogenesis of cerebral actinomycosis. Whether primarily invaded or not, it offers a medium of development most favorable for the parasite. Undoubtedly the thinness of its walls, the numerous cavities and apertures it presents are not strangers to the rapidity of the process. It also contains the dental alveolæ and is covered by buccal mucosa, conditions which make contamination easy. However this may be, it is a bone that can become destroyed quickly, and from here the process invades the meninges. Actinomycosis of the upper jaw is all the more to be feared in relation to involvement of the nervous centres from the fact that therapeutic measures are not brilliant in results.

To sum up it may be said that primary actinomycosis of the nervous centres may exist although this must be very rare. Usually the lesions are metastatic, their origin being in the lung or cervico-facial region. The process may also extend directly to the nervous tissue by the veins, nerve sheaths and various openings in the base of the skull or spine or by destruction of the bones covering the cerebro-medullary axis.

#### SYMPTOMATOLOGY

The relatively few cases so far recorded as well as the richness and variability of the symptoms make it difficult to give a proper description of the symptomatology of actinomycosis of the nervous centres. However, since I gave two pathological forms of the affection, I will give a distinct description of the symptoms of the diffuse and circumscribed forms, but it will be impossible to make all the cases reported fit into one or the other of these headings. Bollinger's patient offered the symptom complex of a cerebral tumor and this was the diagnosis made. In the case I have reported the diagnosis of actinomycotic meningitis was self-evident, but aside from these well-defined cases many are the intermediate ones.

I shall attempt to put a little order in the chaotic state of the symptomatology of the circumscribed form of the process. All that is necessary is to glance at the cases reported to immediately see that certain signs are influenced by the general cerebral irritation, that others, on the contrary, are phenomena due to the localization of the lesion.

Headache occupies the foremost place; it may be extremely intense and be present from the onset. It may not be continued and is subject to remissions and exacerbations resulting from causes often unknown. It is increased by intellectual or physical effort.

As to its pathogenesis it is not as yet clear. It is not always due to hydrencephaly since this is a relatively rare lesion in actinomycosis of the brain. It is true that when hydrencephaly has been absent an abscess has been found which might increase the intracranial pressure. Martin states that in his case the convolutions around the actinomycotic lesions were flattened to some extent, a fact which seems to indicate an increase in volume provoked by the pathologic products. Orloff does not admit, as do most observers, that the pain is due to the special sensibility belonging to the cerebral tissue and states that diseases of the brain are not in themselves painful and pain does not occur until the process has involved the meninges.

This hypothesis agrees well with the fact frequently noted that headache only appears after vomiting and other cerebral symptoms have developed. It also explains the painful phenomena in cases in which no marked intracranial pressure has been found. Unfortunately, the symptom headache is referred to without much detail in most of the reported cases of cerebral actinomycosis. Besides the fact that the actinomyces usually involves both the brain and the meninges—therefore increasing the causes of headache—it may be that the fungus imparts to this pain a special stamp. At least it would be well had the writers stated whether the headache was diffuse or local as this would be important both for diagnosis and treatment.

A symptom which almost always accompanies headaches is vomiting. In several cases these symptoms occurred together. In others it arose before the headache. It is mentioned but without further detail, although Bollinger and Orloff state that it was irregular and that no cause could be found to explain it. If the patients became tired or resumed their ordinary work all the symptoms increased, especially the vomiting. Bollinger states that his patient had previously suffered from dyspeptic symptoms, while Orloff refers to temporary loss of appetite during the evolution of the process but that before the development of the actinomycosis the patients never vomited, and he is certain that this symptom was due to the presence of the parasite in the nervous centres.

Nevertheless, the numerous cases in which this symptom was noted do not elucidate its pathogenesis. The gastric disturbances might be explained by Hayem's theory, which maintains that even in instances of nervous vomiting, this is due to a reflex dependent upon disturbances of gastric chemism. But these disturbances have been found wanting in some cases. Therefore, the pathogenesis of this symptom still remains to be explained.

All the cases in which there were symptoms of cerebral localization of the fungus have at a given time presented evidences of intellectual weakness, but this has not been permanent and presents remissions. Nevertheless, the more the process increases, the less marked are the remissions, and a time comes when loss of consciousness is almost absolute. This symptom is not noted in all cases. In one instance in which it was very marked, the autopsy revealed a rather considerable internal hydrocephalus and it is possible that variations in this hydrocephalus may not have been without influence on the intellectual state of the patient.

The fluid in reality does not greatly decrease so that all parts of the brain, and those presiding over psychic operations in particular, are compressed and finally undergo disturbances which render them physiologically useless. It has also been shown that an increase of the cerebrospinal fluid is not the rule in actinomycosis of the nervous centres, and on the other hand, when the brain lesions are metastatic, as is usually the case, the patient is already worn out by long months of suppuration or by visceral actinomycosis, so that death occurs soon after the cerebral symptoms occur.

The eye is also involved. The optic nerve possesses a sheath which is in communication with the subarachnoid spaces and when tension is increased there will be symptoms in the papillæ in the form of œdema. Double optic neuritis was found in one instance, but I know of no case of atrophy of the optic nerve provoked by lesions of actinomycosis occupying the centres of origin of this nerve.

Concussions have been met with even when there was no localization of the actinomycosis in the motor centres. It is a symptom of an irritative order.

The existence of vertigo has likewise been observed. The vertigo has nothing distinctive, but we do know that it is neither optic vertigo nor cerebellar vertigo. It is probable that this phenomenon is due

to hydrostatic cerebral disturbances, the result of the parasitic neoformation.

I now wish to refer to some symptoms, whose importance from the viewpoint of precise diagnosis, as well as treatment, are of capital importance. The motor convulsions are the most commonly involved in actinomycosis, and their lesions have resulted in such distinct symptoms that in at least one case operation resulted in a temporary recovery. I would, however, warn enthusiasts against the very legitimate tendency to carry localization too far, which in other brain affections have resulted sometimes in useless operations. Paralyzes, and the flaccid variety in particular, have only a relative value in localization. However, they are almost always crossed, and clinically a left sided paralysis should lead to a diagnosis of a lesion in the right hemisphere. Jacksonian epilepsy has a much greater value and should localize the lesion in the centre governing the region in which the onset of the convulsions arises. I do not, however, attribute any absolute value to this symptom for obvious reasons.

The first symptoms which reveal the involvement of the motor centres by the fungus are convulsive. They usually affect the type of partial epilepsy. To have any diagnostic value they must offer a distinct type, always beginning in the same muscles and extending to the remainder of the body following a well-defined progression. In Keller's case the diagnosis was verified at operation, the convulsions beginning in the left arm. The lower limb was seized before the face, a condition contrary to what usually occurs. The brachial type has been twice met with and has been seen to alternate with the facial type. This is an extraordinary phenomenon in Jacksonian epilepsy. But if the pathology be considered, it will be recalled that the actinomycetes do not usually give rise to a single abscess; they are multiple and it may very well happen that the phenomena of irritation are produced in different parts in the space of a few days. The crural type has not so far been observed.

Paralyzes are more common than convulsive seizures. The epileptic attack testifies to a reaction of the cells of the gray matter, but they are little by little destroyed by the parasite and a time comes when a paralysis becomes permanently established. In other cases the nervous tissue has not had time to react, it is at once rendered powerless, or the fibres which communicate with the motor fas-

ciculi are destroyed. In these circumstances there will be a hemiplegia which was not preceded by convulsive seizures, as occurred in one of Martin's cases.

The paralyzes caused by actinomycosis are usually extensive; the parasite, which extends like "a spot of oil," rarely gives rise to symptoms as localized as in ordinary cases of brain tumor. Monoplegias are uncommon, at least when the process is not an actinomycoma. In this case they may occur, as witness Bollinger's case in which there was paralysis of the external rectus of the eye.

I would merely mention muscular atrophy present in Orloff's case. It undoubtedly has the same cause as that giving rise to hemiplegia.

In several reported cases disturbances of speech have been noted, and nevertheless, autopsy showed that Broca's convolution was intact. Almost all portions of the brain may be invaded by the actinomycetes. The intellectual zone of the frontal lobe, the occipital or temporal lobes have been transformed into a pool of pus and nevertheless the patients did not present deafness or verbal or psychic disturbances. I believe for that matter, that actinomycosis, like syphilis may provoke all the syndromes resulting from a lesion in some part of the cerebromedullary axis.

I shall not refer to symptoms arising from actinomycosis in the subcortical region; the cortex and white matter are usually involved simultaneously so that symptomatic distinction becomes impossible. Cerebellar symptoms are not mentioned in any of the reported cases although the cerebellum was the seat of lesions in some. This may be due to absence of lesions of the vermis as pointed out by Orloff.

A word should be said of meningo-encephalitis due to actinomycosis. This process undergoes its evolution in a few days, the intellectual disturbances are very pronounced. The brain reacts by a violent intellectual excitement, delirium and hallucinations occur and the clinical picture is that of the symptomatic complex of acute meningitis. Pyrexia is high and motor disturbances are present. Opisthotonus was observed in the case reported as well as carphologic movements of all the muscles. The process is subject to remissions and a marked one occurred in my case.

## PROGNOSIS

Actinomycosis of the nervous centres is serious, whether it assumes the acute or chronic form, and death is the inevitable outcome. When the nervous centres have become invaded by the fungus, remissions in the symptoms do occur, these being due to phenomena of organic tolerance. When the process affects the acute meningitic form, death takes place in a few days. The neoplastic variety may last several months or even a year.

## TREATMENT

This may be summed up in a few words: Massive doses of KI and surgical interference according to indications.



## THE TREATMENT OF ACTINOMYCOSIS WITH POTASSIUM IODIDE

By D. G. BÉRARD, M.D.

Navy Medical Service, Bordeaux, France

---

THE experiments undertaken in 1893 by a commission appointed by the government to ascertain the therapeutic value of the internal exhibition of KI in bovine actinomycosis were carried out in 185 animals, of which 131 were cured. As the disease is common to cattle and man, Nocard, on April 26, 1892, proposed applying the KI treatment, which had already given such brilliant results in animals, to human beings afflicted with infection by the fungus. In this same year two Dutch physicians, Van Iterson and Salzer, resorted to it in four cases with four successful results. The treatment was then essayed by Buzzi and Galli Valerio in Italy; Hersen, in Austria, and in France by Poncet, Netter, Meunier and others.

Actinomycosis has been regarded as being closely related to two parasitic diseases, namely, botryomycosis and Madura mycetoma and for a long time the parasite of Madura foot was confounded with the actinomyces because the differences between them were few. But the internal exhibition of KI has no action upon the latter affection.

The results of the American commission were final as to the excellent results obtained in the treatment of bovine actinomycosis and if we go over the literature of the last twenty-five years we may conclude that its results are satisfactory in man. The diagnosis of actinomycosis is more difficult in the human species because the lesions are not characteristic like the wooden tongue and the actinomycotic osteosarcoma of cattle. But in most cases a microscopic examination has revealed the true nature of the process in man.

Success is fairly constant when KI is administered internally, no matter what may be the site of the lesions—excepting the nervous centres—nor what may be the form affected by actinomycosis. In many instances surgical treatment applied before the iodide treatment was given was fruitless in results. It may be objected that actinomycosis—even in an advanced degree—may be recovered

from spontaneously, and this is unquestionably true. Schlange, of Berlin, after an experience with thirty cases of the disease, admits that spontaneous recovery does occur by way of suppuration. Poncet, of Lyons, in a letter to the writer, mentioned the case of a female under his care for actinomycosis of the face and lungs who made a spontaneous recovery. But all these instances—besides being very rare—require at least a year or more and cannot in any way compare with those treated with KI.

Let me relate one case, among others, from my notes, to show the really surprising results obtained from KI; not that I would convey the idea that all cases are so successful, but simply to show what can be done in many.

Male, *æt.* thirty-six, farmer. Three years ago the patient, whose dentition was very bad, noticed a swelling on the left side of the lower lip. On August 1, five or six hard papules seated on the inner surface of the lower lip were discovered, vaguely resembling lichen planus. At about the middle of the cheek was a sinus which gave issue to a viscous fluid. There was also an oval tumor, measuring six by two centimetres, lying astride the lower jaw, adherent to the bone by its edges and limited on each side by a furrow which seemed to be hollowed out in the lower jaw. A little below this tumor was another the size of a bean, immovable and adherent to the subadjacent structures.

The patient also complained of attacks of colic for about a year past which varied in duration from one to six hours. He is also very constipated.

*Diagnosis.*—Actinomycosis of lower lip, cheek and jaw.

*August 23.*—After twenty days treatment with KI the patient's general condition was greatly improved; the attacks of colic have not returned. The infiltration of the suprahyoid region has completely disappeared; skin is soft. The tumor astride the lower jaw has considerably diminished, and the sinus has closed.

A few days later recovery was complete and has remained so. The colics and constipation have ceased entirely. These notes were made at the end of November, therefore three months after treatment had been stopped.

The intestinal attacks presented by this patient, likewise the severe constipation, offer considerable interest because they pointed

to the onset of intestinal actinomycosis. It is difficult to be positive on this point, but what would seem to confirm this possibility is the marked influence of the KI, since they disappeared.

It may be remarked that in at least 50 per cent. of the cases in man there have been sinuses or suppuration, and KI easily effected a cure. As to surgical interference, I am disposed to believe that in cases other than intestinal actinomycosis and pleural infection by the fungus, a free incision will suffice. If KI treatment fails, as of course it will in some cases, more extensive surgery will be required.

In considering the length of KI treatment one must distinguish between the time necessary to effect a cure of the lesions and the time during which the exhibition of the drug is to be continued after cure has taken place.

In the bovine race according to statistics published by veterinary surgeons and in those cases where the process is confined to the tongue and adjacent soft structures, a cure is obtained by KI in a fortnight or even less. When the lesions are more extensive, involving the parotid gland or bone, from one to two months will be required.

It would seem that in man the affection persists more or less according to the nature and site of the lesions. A cure is generally obtained in from two weeks to twenty-five days on an average, when the face, thorax or other soft structures are involved. Lesions of the submaxillary region and bone usually require from one to three months, with or without some surgical procedure.

Recurrences are not common and when they occur it is usually within a week or ten days after apparent cure. They are undoubtedly due to stopping the exhibition of the drug too soon. They are, however, benign and quickly give way to the treatment when again resumed.

I shall now briefly consider the period during which KI should be continued. In the bovine race from eight to twelve grammes, given in two doses, is the ordinary daily dose. Fifteen grammes have sometimes been exhibited. As soon as a certain degree of improvement is noted, the daily dose may be reduced to five grammes. Some observers always prescribe the same dose during the entire duration of the treatment; others employ progressively increasing and decreasing doses. All these methods appear to be equally good, and iodism is produced in all.

In man, clinical observation shows that the average dose employed is two grammes daily. Some, like Dubreuilh, prescribe three grammes; Netter gives six grammes daily and then progressively diminishes the dose to two grammes. I believe from personal experience that medium doses—two to three grammes—are sufficient because the process appears to take a little longer to be cured in man than in animals, so that the exhibition of the drug must be continued much longer in the former. For this reason one must not try to obtain symptoms of violent iodism, as is done in the case of cattle.

KI treatment is well borne and all that the physician need do is to watch for the ordinary symptoms of iodism which are never very serious. The drug should be continued for some time after the symptoms have disappeared.

When abscesses form they should be opened and usually a simple incision will suffice as I have already remarked.

Years ago I carried out experiments in respect to the action of KI on cultures of the actinomyces and like all other observers I found that its presence in the culture media, even as high as 5 per cent. had no effect upon the fungus and had no inhibitive effect.

The forms presented by the actinomyces are numerous. Does this polymorphism conceal different varieties of the fungus? I believe that it simply depends upon the conditions of culture. In any event, even should varieties be found to exist that are sensitive to KI *in vitro*, it is none the less true that a cure may be obtained when the fungus does not undergo this action in laboratory experiments.

Since KI treatment does not cure by direct action on the actinomyces, it therefore must act upon the anatomical elements—the tissues. I consequently prefer to admit that if KI does not act directly on the parasite, it proceeds in a round-about way, by modifying the tissues involved, perhaps by increasing the resistance and this by an intimate mechanism which escapes us. In closing I would say that regardless of the excellent results usually obtained, KI cannot be regarded as a certain specific of actinomycosis.

# Clinics

---

## ON ABSCESS OF THE PREVESICAL SPACE AND UMBILICUS, WITH SPECIAL REFERENCE TO THEIR ORIGIN FROM CYSTS OF THE URACHUS, AND REPORT OF A CASE SIMULATING URACHAL CYST

By DAMON B. PFEIFFER, M.D.

Associate in Surgery, University of Pennsylvania.

---

ABSCESSES and cystic formations which are found in the deeper layers of the anterior abdominal wall below or in immediate relation to the umbilicus possess unusual interest from the standpoint of etiology and treatment. Before the recent development of surgery and the rational early treatment of infective conditions, such abscesses were not uncommon and many extraordinary cases are recounted in the literature up until about thirty years ago, since when reports have become increasingly rare. However, there still exists considerable doubt concerning the origin and mode of spread of certain of these abscesses. The following case is of interest and importance in this connection.

A. P., negress, aged 17 years was admitted February 7, 1921, to the Hospital of the University of Pennsylvania.

She complained of pain and swelling in the lower abdomen. In January, 1920, she first noticed soreness in the left lower abdomen. In two weeks this passed away. In November, 1920, the same condition returned and lasted about three weeks, again followed by improvement. After this attack there was some persistent soreness below the navel. At this time she had night sweats. About January 1, 1921, pain reappeared more especially in the right lower abdomen and a swelling formed in the midline above the symphysis. About three weeks later the umbilicus began to protrude and became tender.

Some loss of weight had occurred. There were no pulmonary, vesical or gastro-intestinal symptoms.

The patient was well developed and fairly nourished. The head, neck, chest and extremities presented no noteworthy abnormalities.

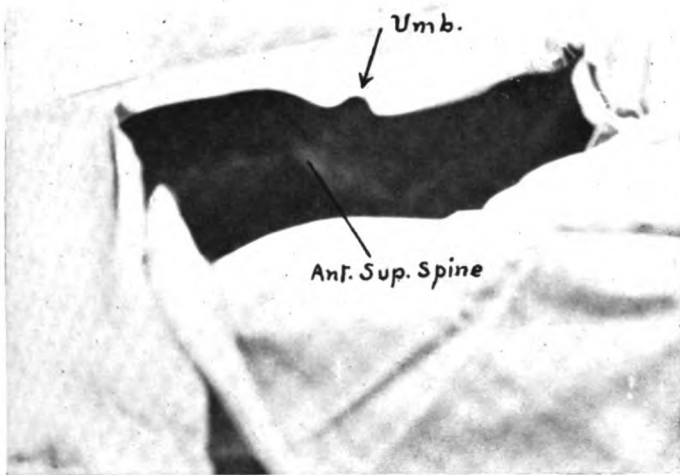
Below the umbilicus a symmetrical oval prominence was seen rising from above the symphysis. Its long axis was exactly in the midline and laterally it extended almost to the linea semilunaris on each side.

Its upper pole extended to one and one-half inches below the umbilicus. From this point upward to the navel the tissues were normal. The umbilicus itself protruded forming a conical elevation about one inch in height like an umbilical hernia. The skin overlying both prominences appeared normal but the slightest pressure caused pain. They were soft and clearly cystic. The percussion note over the most prominent portion was slightly tympanitic and the crackling sensation of gas could be elicited by palpation as well as a fluid wave by tapping. Gas and fluid could be made to pass from the umbilical to the lower swelling and vice versa. It was evident that this passage did not take place through the subcutaneous tissues but through a deeper communication. Rectal examination and vaginal examination under ether prior to operation showed a conglomerate mass apparently comprising the uterus and appendages which was coextensive with the abdominal tumefaction. This extended backward and downward, but did not extend to or involve the floor of the cul-de-sac. It was moderately tender and immobile. The pulse varied between 90 and 110 per minute and the temperature between 98° and 102° F. The urine was normal. Blood examination showed Hgb., 50 per cent.; R.B.C., 3,860,000; W.B.C., 19,000. Blood pressure was 108 systolic, 78 diastolic. The Wassermann was negative.

A tentative diagnosis of infected cyst of the urachus was made and cystoscopic examination by Dr. F. E. Keene showed the bladder and ureteral orifices to be normal, but in apparent confirmation of the diagnosis there was seen protruding from the wall of the summit of the bladder a teat-like projection, slightly inflamed, situated just where the urachus would normally enter the bladder. There was, however, no discharge, nor could any orifice be found.

Operation, February 9. A midline incision over the larger prominence at once enters an abscess cavity filled with a small amount

FIG. 1.



Lateral view showing scaphoid epigastrum, protruding umbilicus and hy-pogastric prominence of the subcutaneous abscess.

FIG. 2.



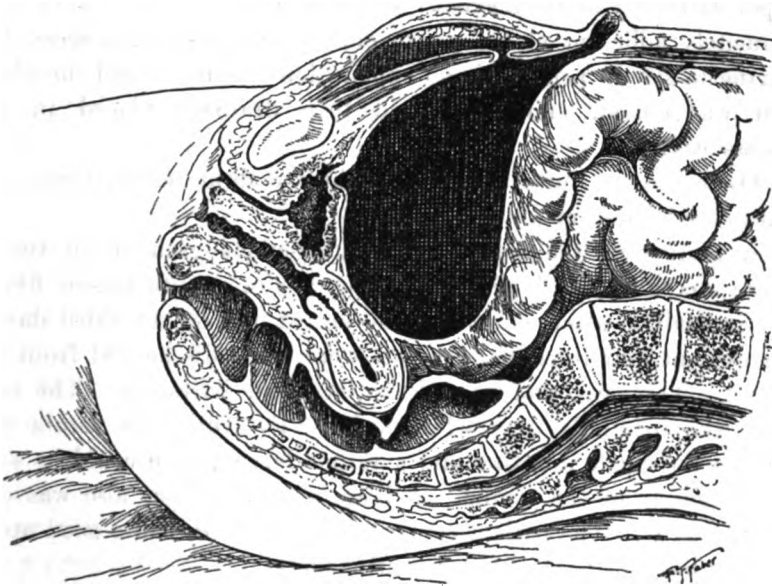
Cystoscopic appearance of hillock seen at the summit of the bladder at the vesical termination of the urachus.

Digitized by Google



of gas and considerable non-odorous reddish-brown purulent-looking material. This was mopped away and the linea alba and adjacent rectus sheaths exposed. An erosive and solvent action by the fluid or its contained infection was evident upon the aponeurotic covering of the muscles which was thin and in some places completely eaten away exposing the red and apparently viable muscle fibres of the recti. This subcutaneous abscess did not communicate directly with the umbilicus.

FIG. 3.



Schematic representation of the site of the abscess.

About midway between the navel and symphysis just to the left of the linea alba, pus could be seen exuding from a small sinus. A probe passed through this entered a cystic expansion below. The linea alba was then divided longitudinally opening a cavity containing about 500 cc. of pus very similar to that noted above but not quite so bloody. The cavity communicated above beneath the rectus sheath with the umbilicus. From this point it passed downward anterior to the peritoneum, expanding gradually, funnel-like, until it reached the brim of the pelvis, when it dipped downward into the pelvis forming a cyst-like expansion, the anatomical boundaries of which could not be completely recognized. It abutted upon the sum-

mit of the bladder, but no structures could be traced merging with that viscus other than the lining membrane which was everywhere similar. This was irregular in contour, soft, friable and grayish with small gray fibrinous masses adhering. There appeared to be no definite cyst membrane, and attempts to remove the grayish surface led to lively oozing. The finger could be passed above beneath the abdominal wall into the umbilical cavity. The pus was sponged away and in attempting to define the exact limits of the abscess the finger passed without resistance into the abdominal cavity at the upper extremity of the abscess. It was seen that the intestines were intimately adherent to the peritoneum underlying the abscess, but fearing to set up peritonitis no exploration was made and the abdomen was closed, and gauze and tube drainage placed in the abscess cavity.

Owing to an unfortunate oversight the fluid removed was not sent to the laboratory for examination.

A section of the wall showed tuberculous granulation tissue. There was no trace of epithelial lining, nor of smooth muscle fibres. The immediate aftercourse was uneventful, but on the third day it was evident that intestinal contents were being discharged from the wound. This was profuse, bile stained and irritating. The contents were partially digested material. Evidently the fistula was high in the small intestine and a rather large orifice could be seen in the bottom of the wound. A large amount of pus also was discharged daily. The patient continued to feel well, had a good appetite, but lost weight slowly and continuously.

A second cystoscopic examination of the bladder six weeks after operation showed that the intravesical projection above noted had disappeared and no trace of it could be seen.

The cavity gradually diminished in size and the discharges of both pus and bowel contents were considerably reduced but the temperature remained unaffected showing the same diurnal variations as before operation. As emaciation progressed it was felt that the loss of nutriment was a serious detriment and the abdomen was opened under local anæsthesia in order to sidetrack the fistula by a lateral anastomosis between the bowel above and below the opening. This was accomplished without difficulty and the opportunity of inspection at this time showed a moderate number of tubercles stud-

ding the peritoneum and some caseous glands in the mesentery. The pelvic organs were unrecognizably fused with the wall of the lower extension of the suppurating tract, and as noted at the primary operation many loops of small intestine were also densely adherent.

This operation was partially successful in diverting the intestinal contents. There was but little loss, the bowels moved normally, but weakness and loss of weight progressed until death, June 25, 1921. No autopsy was obtained but it is felt that the operative and other findings permit a satisfactory critique of the condition.

Comment on this case will advantageously be postponed until consideration is given to related conditions.

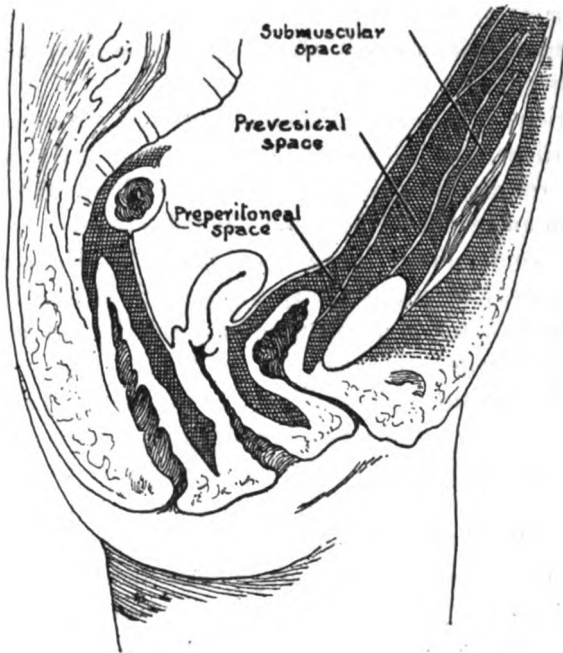
The various situations and characteristics of the suppurating and cystic conditions at or about the umbilicus have led to a number of anatomical researches which have as their object the explanation of the origin and cause of these cyst-like collections.

Notable contributions have been made by Heurtaux, Joüon, Henke, Leusser, Pinner and Charpy, as a result of which the classical space of Retzius, which is bounded in front by the recti muscles, behind by the peritonium, below by the bladder and symphysis and above merges with the peritoneal space in the region of the umbilicus, is found to be made up of a number of compartments and to contain structures that influence the localization and direction of spread of contained fluid. The results of these studies and observations do not agree in all particulars, and it is probably that nature in laying down these fascial planes which are delicate and not too well defined may present many individual variations. In general it seems, according to the summary of Ledderhose, that each of the two recti is provided with a separate sheath which is not interrupted in its posterior aspect. Below the semilunar folds of Douglas, the transversalis fascia, which lines the entire anterior abdominal wall, assumes the role of the posterior muscle sheaths and inserts into the symphysis and pubic rami. It separates the recti muscles from the peritoneal cellular tissue. The space, occupied chiefly by loose areolar tissue, is further divided by a fibrous layer, the fascia propria of Velpéau (feuillet prévésical, Charpy) which presents itself in a membrane-like thickening of the subserous cellular tissue surrounding the vesico-umbilical ligaments and the urachus and several centimeters below the folds of Douglas separates from the transversalis fascia and

passes down over the anterior surface of the bladder to unite with the pelvic fascia.

We have, therefore, in the hypogastrium between the recti muscles and the peritoneum three spaces occupied normally by loose areolar tissue; (1) between the muscles and their posterior sheaths, namely, the submuscular or suprapubic (Leusser) space lying above the pubic bone, bounded in front by the posterior surface of the recti and behind

FIG. 4.



Sagittal frozen section of a female cadaver after artificial infiltration of the tissues with water by arterial injection under pressure of about 10 feet.

by the transversalis fascia; (2) between the posterior surface of the rectus sheaths and the fascia propria of Velpeau, namely the prevesical space proper. According to Charpy, a further space (3) exists between the fascia of Velpeau and the peritoneum, the preperitoneal space in a restricted sense.

Inferiorly, the posterior sheaths of the recti become thin and here the lower part of the submuscular space can communicate through small apertures with the prevesical space. The rectus sheath spaces are further subdivided by the tendinous inscriptions and firm adhe-

sions at their lateral borders preventing inter-communication between anterior and posterior sheath spaces. The transversalis fascia is continued beneath the posterior surface of the umbilicus and the fascia of Velpeau is inverted into the structures of the umbilicus together with the vesico-umbilical ligaments, losing its identity above this point.

The distensibility of the preperitoneal and prevesical spaces and the elasticity and looseness of their cellular tissues under normal circumstances can be appreciated by anyone who has ever seen the intra-abdominal relations of a distended bladder. The possibilities of distension of these spaces by pathological fluids or cysts will be similarly understood. It must be mentioned also that from the work of Heurtaux, Joüon and Fischer it appears that there is a heart-shaped space lying base upward just below the umbilicus. According to Fischer it is situated between the peritoneum and Velpeau's fascia and is divided into two parts by a complete median septum.

The role of the urachus in cyst and abscess formations in this location is still not entirely settled. It is well known from the researches of embryologists that the urachus is the remains of the fetal allantois. This tubular structure which is given off from the hindgut in the first month of fetal life, makes its way out into the body stalk, but in the human, unlike its behavior in certain animals, it never attains great size. As development proceeds, it is found passing out of the coelom in the substance of the umbilical cord in company with the omphalomesenteric duct and the umbilical vessels. Its extrafetal course is short and under normal circumstances becomes obliterated before the end of fetal life. Intraabdominally at its lower extremity it joins the hindgut, from which it was originally formed, in the common chamber of the cloaca. By the development of a longitudinal septum, the anterior and posterior portions of the cloaca are separated, the latter developing into the rectum and the anterior portion being concerned with the formation of the urogenital tract. The urachus, therefore, remains in communication with the summit of the bladder. Obliteration normally begins in the later months of fetal life, proceeding from the umbilicus downward, but as in all analogous embryonic processes, this process varies in point of time and completeness. It may completely fail, giving rise to the interesting anomaly of completely patulous urachus. This

may manifest itself at birth by the escape of urine from the umbilicus and require surgical treatment to effect closure, or it may close spontaneously soon after birth and remain continent until back pressure or infection or both induce reopening, which may not take place until very late in life.

In other cases the umbilical end of the urachus may remain patulous for a short distance, ending blindly by reason of obliteration of the lower segment. It seems that this upper blind sinus is an uncommon anomaly because of the strong tendency of the umbilical end of the canal to close first. Probably also the possessors of the abnormality are commonly not aware of the fact unless irritative processes are set up in the sinus, due to retained cellular detritus, or to accumulation of filth in the old and uncleanly. Under these circumstances there may be an irritating and foul secretion from the umbilicus. Abscesses or cystic dilatation of the sinus may arise and concretions have been found.

More frequent than persistent sinus at the upper end of the urachus is a failure to close at the vesical end. According to Luschka, Wutz and Binnie, this may be regarded as the normal condition since it occurs in the majority of individuals and persists throughout life. In most cases, however, the opening is small and the canal short, requiring special methods of preparation for detection. Wutz, in the examination of seventy-four cadavers of all ages, states that if one observes the inner surface of the mucous membrane of the bladder, in the majority of cases, providing it be somewhat spread out, one can detect a funnel-shaped indentation and at the summit of this little funnel a small opening. In 51 cases (69 per cent.) a bristle could be passed into the lumen. In 32 cases the depth varied from 2 mm. to 6 mm., in 19 cases from 1.1 cm. to 4 cm., and in 21 cases a very evident pit could be seen at the point of attachment of the urachus. In only two cases, (2.7 per cent.) was there no trace whatever of the original communication. Wutz pointed out that at the entrance of the urachus there is ordinarily a transverse fold which makes it difficult to pass a probe, and during life usually prevents the entrance of fluid. Doran has formally dubbed this structure Wutz's valve.

Binnie reports Clendenning's study of the urachus in 16 adult cadavers and in 6 fetuses. In 7 adults and 5 fetuses, the bladder showed a distinct diverticulum from 1 cm. to 2 cm. in depth at the

summit where the urachus is attached. In an adult, there was a slight projection instead of a diverticulum. In 8 adults and 1 fetus the dome of the bladder was smooth. The urachus was usually adherent to the belly wall, but in one case (diabetic with frequent retention of urine) it was not close to the parietes, but lay between loops of small intestine. Binnie attributes this condition to a persistence of the fetal state where the urachus possesses a meson. Its bearing upon the possibility of an intraabdominal situation of an urachal cyst as distinguished from the preperitoneal location is of some importance.

In addition to the open lower end of the urachus, Luschka found segments of the canal which were unobliterated though closed above and below. Wutz and others have confirmed this finding. Such channels may contain fluid, or at times solid or semi-solid materials of varying composition. They may be straight globular, fusiform, or tortuous and often present diverticula which may themselves become separated from the parent canal. Although no clinical report of cyst of the urachus had been placed on record at the time, Luschka perceived that in these structures were all the elements necessary for the formation of cysts and voiced his belief in the probability of their existence. He described accurately the finer structure of the urachus, giving criteria by means of which urachus cysts could be recognized. The epithelial lining consists of a single layer of cells or of several layers of stratified or transitional epithelium resembling that of the bladder. Next comes a structureless basement membrane and a connective tissue layer. Outside is a layer of smooth muscle, the fibres of which run irregularly but chiefly in a longitudinal direction.

Following this suggestive paper, reports of cysts of the urachus observed clinically or at post mortem began to appear in the literature, notably by Wolff, Atlee, Roser, Hoffmann, Freer, Lawson Tait, Robinson, Wutz, Doran and many others. Some of these cysts were small, discovered accidentally and their origin was certainly established. Many, and in fact, practically all of clinical importance lacked more than one of the histological criteria established by Luschka to which Wutz added extraperitoneal location in the tract of the urachus. Wutz admitted the validity of none of the larger cysts reported clinically up to his date, 21 years after Luschka's communication. He accepted the case of Walter, reported in 1800 and here

pictured (Figure 6). The dimensions of these structures are not impressive. The first large cyst had a length of 2.5 cm. and a breadth of 1.6 cm. The two associated cysts were somewhat smaller.

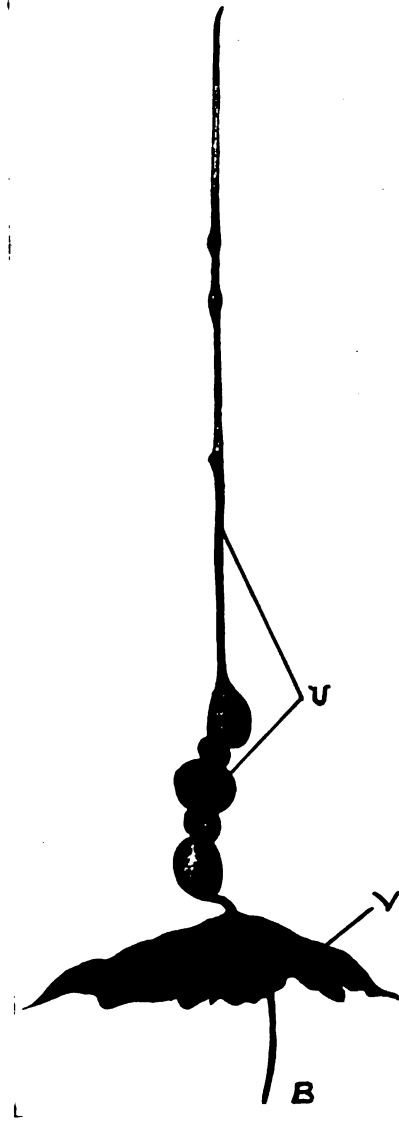
Doran (1898) reported the case of a woman aged 59 years who had suffered for nine months with lower abdominal pain and a mass lying slightly to the left of the midline. Operation showed an infected preperitoneal cyst containing about a pint of dirty greenish-brown fluid and shreds. Histological examination of the cyst wall showed no epithelium, but it contained fibrous tissue and much smooth muscle. He considered the diagnosis established and in his rather comprehensive review of the subject, he relaxes somewhat from the rigid requirements of Luschka and Wutz, but still can find no veritable cyst of the urachus larger than that of Bryant, which was the size of a cocoanut. He supports Wutz in ruling out the so-called allantoic cysts reported by Tait, regarding them as examples of tuberculosis or encysted peritonitis. He is disposed to admit, however, the case reported by Douglas which contained 25 pints of clear, pale green fluid containing a few flocculi. In this case the cyst wall was removed and consisted of fibrous tissue showing neither epithelium nor smooth muscle. It had no special area of attachment even to the bladder. The diagnosis rests solely on its situation.

Weiser, in 1906, collected 86 cases to which he added 3, under the title of cysts of the urachus, including almost all that had been reported in the literature to date. He constructed a valuable table for reference and made a brief analysis of the clinical features.

Unfortunately for a correct understanding of this condition he subjected the cases to no searching criticism. Many are certainly not urachus cysts. Not more than 10 can stand rigid criticism; 27 are examples of cystic sinus and the remainder range from probable to doubtful. He includes the largest and oft quoted case of so-called cyst of the urachus on record (Hoffmann's) which after death and numerous previous tapplings was still found to contain over fifty litres of fluid. He includes this same case a second time, crediting it to Freer, who took the liberty of using it without giving his source. Other authors have fallen into this error, thus giving the impression that there were two cysts of the urachus reported to have contained over fifty litres of fluid, while in truth, following Wutz, all critical writers have rejected Hoffmann's case as resting on insufficient proof

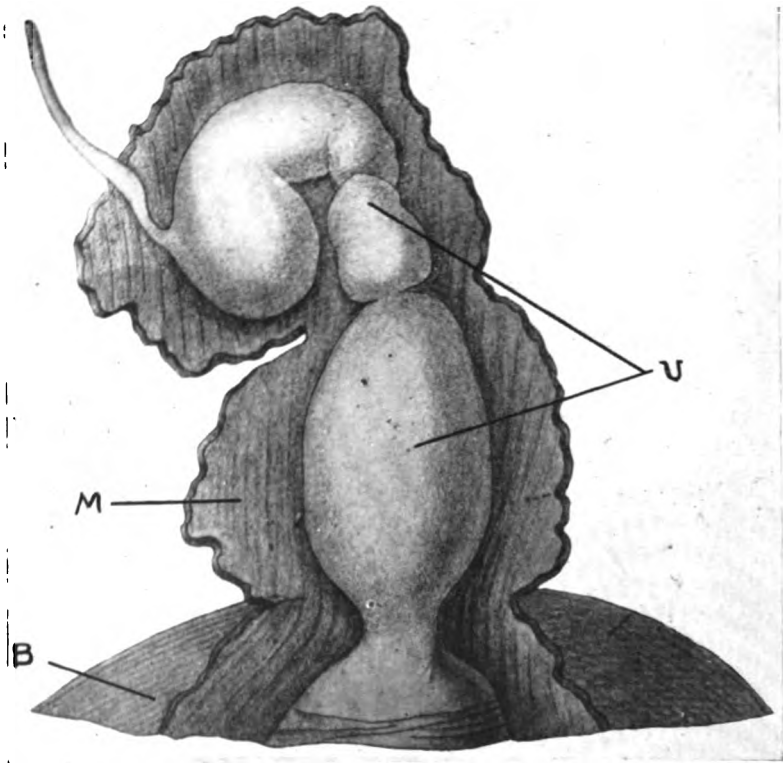


FIG. 3.



U. Urachus with cystic dilations at vesical end. V. Bladder. B. Bristle inserted into orifice of urachus. (Wutz.)

FIG. 6.



U. Urachus cyst. The muscular coat, M, has been separated from the mucosa and reflected. B. Bladder (after Walter).

and without clinical parallel except in conditions which were certainly not urachus cysts.

In the literature since 1913 I have found 15 cases formally reported as urachus cysts, 2 as tuberculosis of the urachus and about 20 each of tumors (chiefly carcinoma) of the urachus, and of various forms of persistent sinuses or cystic dilatations of patent sinus. Of the 13 cases reported as cysts, 7 may be considered as positive (Doran, Means, Delore and Cotte, Chavannaz, Weber, Bonzani, and Moore). Five are probable or possible (Baldwin (2 cases), McDonald, Tricot, (ifuentes). One (Jacoby) is not in any way substantiated. Two reports (Gramén, Aleman) were not available in this country.

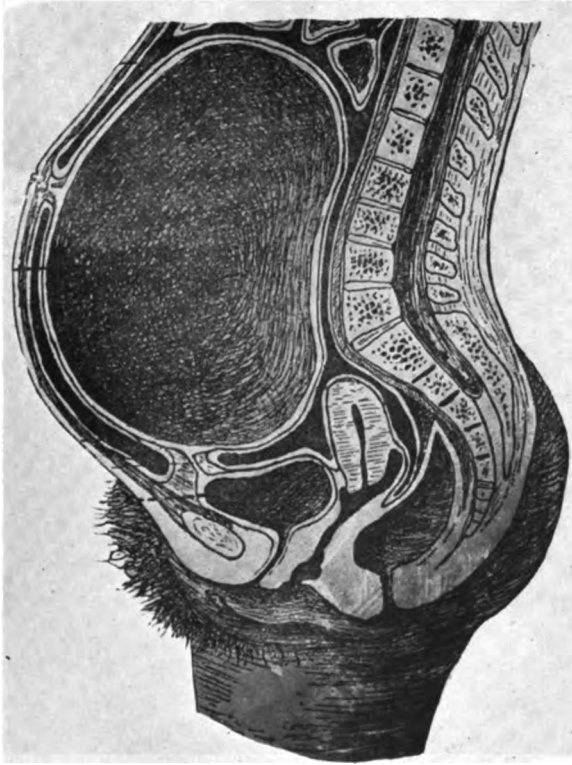
The largest cyst was Delore and Cotte's, which distended the abdomen to about the size of a nine month's pregnancy. Chavannaz's cyst measured 10 cm. in diameter. Delore and Cotte's was unique in being intraperitoneal. Binnie's observation of a meso-urachus attests this possibility. Means's cyst was sessile rather than intraperitoneal. There is no evidence that any of the positive cysts was infected. The probable, possible and inadmissible cysts, however, were suppurating. This is the feature which makes it difficult to make a differential diagnosis in the given case between a suppurative collection and a possible urachus cyst in this location and which makes it impossible to determine in many cases reported in the literature as urachus cyst whether its author was in reality dealing with an instance of that condition and indeed makes even the most careful biopsy or autopsy too often inclusive, since it is readily conceivable that infectious processes once established could readily cause necrosis of all epithelium, transform the wall into fibrous tissue only, and even break out into adjacent structures leaving the confines of its walls and thereby escaping all trace of its origin. That urachus cysts do become infected there is no doubt. The stickler Wutz himself, in two of his autopsies, found small infected cysts, in one instance associated with death from prostatism, in another, from appendicular peritonitis. Both were clinically unimportant and would have been overlooked but for Wutz's searching inquiry into this special field. Still the observations are more than suggestive and while the number of reported and established cysts of the urachus is small, it is probable that at least some abscesses in this location

centrally in the abdomen can be outlined and soon the navel protrudes, fluctuates, shows signs of inflammation, and either ruptures spontaneously or is incised, permitting the escape of a quantity of thick greenish pus, containing the pneumococcus. Fecal fistula rarely occurs and recovery is often rapid and complete.

Still another group, formerly not infrequent, is that in which after puerperal or other pelvic infections, an abscess works its way into the space leading to the umbilicus, finally to make its escape at that point. The advance of pelvic surgery makes this occurrence seldom nowadays. If we were to tabulate all the conditions that have terminated in subumbilical abscess and fistula, it would sound formidable indeed, including not only appendicitis, peritonitis, echinococcus disease, but also cholecystitis, hepatic abscess, empyema and lung abscess and doubtless many others. These, however, belong to the curiosities of medicine.

The interpretation which I place upon my case therefore is this. The antecedent lesions were tuberculous salpingitis and peritoneal tuberculosis. By adherence, ulceration and contiguity the prevesical space was infected and a preperitoneal cold abscess formed. Later the pus perforated the fascia, emerging subcutaneously through the sinus noted at operation. Still later it reached the subumbilical space and was guided into the navel by the anatomy of the fascias and embryonic structures which converge to this point. The intestines naturally adhered to the inflamed peritoneum which confined the abscess on its inner surface and the erosive process was responsible for the fecal fistula which appeared so promptly after operation. The curious protrusion into the bladder at the site of the lower end of the urachus, which was noted at the first cystoscopy, may be accounted for just as the protrusion at the umbilicus, the upper end of the urachus, is explained by assuming that a remnant or vestige of the urachus was present in this individual, as Wutz has shown to be true of the majority of normal individuals. Pus travelling in the direction of least resistance might easily find here, either the thinnest portion of the bladder wall which it pushed forward into the cavity of the bladder, or probably, though less probably, a small intravesical cyst of the urachus was actually present which became infected and bulged into the lumen. In our view of the matter this is a more logical explanation of the case than to assume that a previously sterile

**FIG. 7.**



**Delore and Cotte's case of intraperitoneal cyst of the urachus.  
Schematic.**



cyst of the urachus became infected with tuberculosis and by enlarging and extension set up the lesion found. Yet many reported cysts of the urachus rest upon assumption no less perilous.

It appears certain that in view of the extreme rarity of known cysts of the urachus, a positive clinical diagnosis presents a great hazard and that the greater frequency of suppurative conditions of other origins which may make their way to the umbilicus for discharge, owing to the anatomical peculiarities of the spaces there converging, places the burden of proof on those who would explain such preperitoneal collections as infected urachus cysts.

### BIBLIOGRAPHY

- ALEMAN: *Hygiea*, Stockholm, 1916, lxxviii, 952.  
 BALDWIN: *Surg. Gynec. & Obstet.*, June, 1912.  
 BONZANI: *Pensiero med.*, 1911, 1,365.  
 BRIDGON AND ELIOT: *Med. and Surg. Rep. of the Pres. Hosp. N. Y.*, iv, 1900, 30.  
 BINNIE: *Jour. Amer. Med. Asso.*, 1906, xlvii, 109.  
 CAMEBON: *Proc. Roy. Soc. Med.*, Feb., 1912, v, No. 4, 123.  
 CIFUENTES: *Rev. de med. y. cirurg. Prac.*, Madrid, 1915, cxii, 342.  
 CHAVANNAZ: *Ann. de Gynecol.*, 2 série, 12, 1916-17.  
 CULLEN: *The Umbilicus and its Diseases*, W. B. Saunders Co., Phila., 1916.  
 DELORE AND COTTE: *Rev. le Chir.*, 1906, xxxiii, 403.  
 DORAN: *Medico-Chirurg. Trans.*, vol. lxxxi, 1898.  
     *Jour. Obstet. and Gynaec.*, Brit. Emp., ii, 1907, 435.  
     *Lancet*, (London) 1909, 1, 1304.  
 EASTMAN: *Amer. Jour. Obstet.*, 1915, lxxii, 640.  
 FISCHER, H.: *Volkmann's Samml. klin. Vorträge*, n. F. 89 (Chir. 24) 1891-94, 519.  
 FREER: *Ann. Surg.*, 1887, v, 107.  
 GRAMEN: *Hygiea*, Stockholm, 1916, lxxviii, 1460.  
 HENKE: *Deutsche Zeitschr. f. prakt. med.*, 1877, iv, 486.  
 HEURTAUX: *Bull. et Mém. de la Soc. de chir. de Paris*, n. s. 1877, iii, 641.  
 JACOBY: *Urol. and Outan. Rev.*, St. Louis, 1916, xx, 383.  
 LEDDERHOSE: *Deutsche Chir.*, 1890, Lieferung 45b.  
 LUSCHKA: *Arch. f. path. Anat. u. Physiol. u. f. klin. Med.*, 1862, xxiii, i.  
 MORONE: *Pensiero med.*, 1915, v, 174.  
 McDONALD: *Ann. Surg.*, 1907, xlv, 230.  
 TAIT: *Brit. Gynec. Jour.*, 1886-7, ii, 328.  
 WEISER: *Ann. Surg.*, 1906, vol xlix, 529.  
 WUTZ: *Virchow's Archiv.*, xcii, 387.

## MECKEL'S DIVERTICULUM INCARCERATED IN AN INGUINAL HERNIA

By RALPH B. BETTMAN, M.D.

Adjunct Surgeon Michael Reese Hospital, Chicago

---

This case I am about to operate upon is an inguinal hernia in a child (two and one-half years old). The mother has noticed a lump in the right inguinal region practically since the child was born. Two nights ago the child commenced crying and the mother noticed that the lump was larger than she had ever seen it before. She tried to push it back as she had done on several previous occasions but found that she was unable to do so. A physician was called and after a little effort managed to reduce the hernia. He explained to her the nature of the trouble and the danger associated therewith and advised prompt operation.

As you see, the hernia seems to be entirely reduced. The external ring is large and there is a noticeable muscle weakness on the right side as compared to the left. The right cord is thicker and larger than the left, especially as it leaves the external ring. Both testes are in the scrotum.

To prepare a baby's skin for operation I have the interne scrub with soap and water, apply alcohol, then ether, then a three per cent. solution of iodine which is washed off again with alcohol.

Iodine burns the skin of a baby much more readily than that of an adult and when iodine is used it should be washed off again with alcohol. Another precaution, which ought not to need mention, is to guard against the iodine running down the sides or back and drying in heavy brown ridges. When these are allowed to remain a burn is sure to follow. For this reason it is often best to prepare the patient completely before any sterile linen is applied.

I am making my incision as high as possible. I do this for two reasons. In the first place there are more superficial veins cut the lower the incision is carried. This necessitates additional ligatures; it is more difficult to keep the wound free of blood. Then, furthermore, the fewer blood-vessels injured the less the danger of



post-operative pulmonary complications. That a large percentage of these complications is due to emboli liberated during operations is now a well recognized fact. V. Mikulicz, Henle and others in a series of cases have shown a greater percentage of post-operative pneumonias following local anaesthesia than following ether. Cuttler and Hunt in the first number of the *Archives of Surgery* advance much evidence to show that the inhalation of the anaesthetic is not the chief factor in all post-operative pulmonary complications.

The second reason for a higher incision in herniotomy in children is that the further the wound is from the genitals the less chance there is of having the dressings wet with urine.

I have, as you see, encountered few blood-vessels. There is a much firmer fascial covering over the external oblique in infants than in adults.

I am now down to the external oblique and with the back of my knife, with one easy sweep, I clear it down to Poupart's. The external oblique is here split down to the inter columnar fibres, therefore I will use this natural cleavage, cutting through the inter columnar fibres, and exposing the canal.

In order to find the sac I carefully separate the cord near the external ring with anatomical forceps. In congenital hernia, of course, the vas and its vessels lie in intimate relation to the sac, and after having found the sac it is only by means of careful dissection that they are separated. Here we have the sac freed, it is a short sac and does not extend down along the cord for any great distance. The sac is thickened.

I am opening the sac at its very tip, with great care. The sac is opened. I can spread the incision in it with a hemostat, and will now carefully enlarge the opening with a blunt-pointed scissors. Here is an adhesion. It is a firm and fibrous band attached to the wall of the sac. I wonder what it leads to. By tugging on it very gently I see that it is attached to the small intestine. This is probably the loop of bowel which two days ago became incarcerated for a short time. Let us just have a look at the bowel and see if we can find any reason why it should have adhered. This is strange! The band does not seem to lead to the loop of bowel at all, but to a blind pouch of bowel. It looks almost as if an intestinal resection had been done at one time or another and this were one of the closed ends. In

other words, we are evidently dealing with a diverticulum. Let us see how large a diverticulum there is. It has the diameter, contour and color of normal healthy small gut. Here we have the true intestine. There, this shows the condition excellently. Here is a short, firm fibrous band about one cm. in length adhering to the sac wall near the neck of the sac. This leads to a rounded end of gut which has the appearance of normal healthy bowel which is about four cm. long and about one and one half cm. wide, and which comes from the ileum. A beautiful example of a Meckel's diverticulum.

There are no signs of an inflammatory process either old or recent, no signs of injury to the bowel. There appears to be no other adhesions.

What shall we do? Shall we ligate and cut the adhesion to the sac wall, allow the diverticulum to fall back into the abdomen and finish our herniotomy as we intended? Or shall we, bearing in mind the possibilities of diverticulitis, of an intestinal obstruction due to the diverticulum, now that we have the diverticulum nicely exposed, amputate it and then close our hernia?

I am going to cut and ligate the adhesion. Allow the diverticulum to fall back into the abdominal cavity.

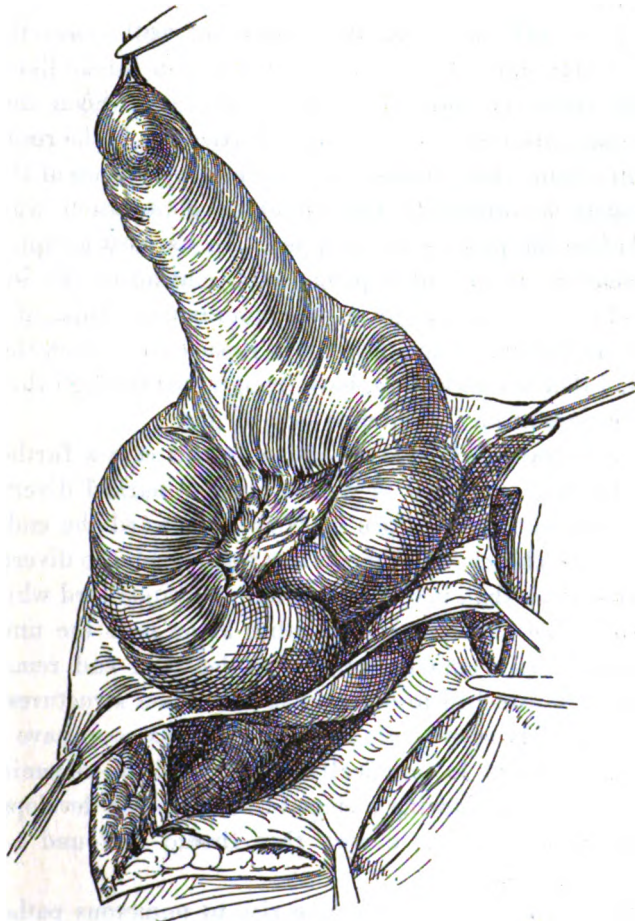
I will explain why I am finishing my herniotomy without invaginating the diverticulum. A Meckel's diverticulum is found in a little more than one per cent. of all cases coming to autopsy. Oviatt, I think, says two per cent. Adami, 2.7 per cent. Now this is not such an infrequency. However, the cases which come to operation for a condition caused by a Meckel's diverticulum are very few and far between. Probably the most frequent complication of a diverticulum is intestinal obstruction and of all cases of intestinal obstruction only six per cent., according to Halstead, are due to a Meckel's. This gives us a fair idea of what a really harmless anomaly a Meckel's diverticulum is.

Technically the removal of this diverticulum would be an easy task but it would have increased even though slightly the danger from infection. Also, there would be the possibility of a hemorrhage. It might happen, that the resultant intestinal scar might give rise to adhesions, a stricture, or act as the site of an intussusception. These sequelæ I will admit are improbable but are they not more improbable

than that this Meckel's diverticulum which is found in about one of every fifty autopsies will cause symptoms?

Meckel's diverticulum is a remnant of the omphalo-mesenteric duct

FIG. 1.



**Meckel's Diverticulum.** The diverticulum is being held up by means of the fibrous band which was adherent to the hernial sac. The loop of gut shown is ileum

As you know, the primary alimentary tract is formed by infolding of the splanchnopleure. This proceeds rapidly at the head and tail-end leaving the middle portion quite open to the yolk sac. This middle portion is the *anlage* of the small intestine. A Meckel's diverticulum always arises from the small bowel. Before the yolk

stalk is entirely shut off the fore part of the alimentary tract and the hind gut have been differentiated. The mid gut or the small intestine consists of one loop at the height of the convexity of which is attached the vitelline duct. On the distal limit of the loop a slight bulbous formation later becomes the cæcum.

During the fifth week, as the abdominal wall closes this duct gradually is obliterated leaving finally but a thin thread-like process which runs from the intestine to the umbilicus. Soon thereafter this thread also disappears. Meckel's diverticulum is the result of an arrest of this obliterated process, and the final appearance of the diverticulum varies according to the extent of obliteration which has occurred before the process was stopped. In its most complete form the diverticulum consists of a patent tube continuing the intestinal lumen to the exterior, usually at the umbilicus. This, of course, means a fœcal fistula. Cases have been reported in which the diverticulum, and then the intestines, have invaginated through this fistula forming a hernia of an invaginated loop of gut.

A less well-developed form, or rather in this case a further well-developed form, is one such as we see here, a marked diverticulum extending from the free margin of bowel. Toward the end of the diverticulum the obliterating process has progressed, the diverticulum tapers—often more than here—and ends in a fibrous cord which may be attached to the abdominal wall either at or near the umbilicus. Often this terminal filament has become detached and remains so. Again it may be attached to other intra-abdominal structures. This attachment probably occurs secondarily. Various cases have demonstrated its attachment to practically all of the intra-abdominal viscera. A type of case showing a stage of still further development is that in which nothing but a very thin thread is found adherent to the small intestine.

A Meckel's diverticulum can give rise to numerous pathological conditions. It may be the cause of an intestinal obstruction, a volvulus or an intussusception. It may perforate; it may become strangulated or incarcerated in every conceivable kind of abdominal hernia. The intestinal obstruction may be caused in numerous different ways. Halstead has divided these cases into two large groups. In the first the obstruction is caused by an unattached diverticulum, when the diverticulum has caused a knot, a loop, a volvulus, etc., etc. In the

second group the obstruction is due to the attachment, the obstruction being caused by loops of gut becoming incarcerated above or below the attachment of the terminal ligament or kinked by torsion, etc., etc.

Let us hope that this little lad will escape all of these unpleasant possibilities, and will lead according to the law of mathematical chances an uneventful life as far as his Meckel's diverticulum is concerned.

While speaking I have completed the herniotomy by the Andrews method without transplantation of the cord. I have used fine kangaroo tendon for my deep suture material. I have taken care to suture the fascia to which I alluded before. I have closed the skin with a very fine catgut, and I am using two half-inch strips of adhesive to reinforce the sutures. In children it is of advantage to use suture material which does not have to be removed.

# Medicine

## THROMBOSIS OF THE INFERIOR VENA CAVA AND BOTH RENAL VEINS \*

By F. PARKES WEBER, M.A., M.D., F.R.C.P.

London, England

THE patient, G.G., aged forty, a man-cook, was admitted to the hospital in September, 1920. In the spring of 1919 he had suffered from a septic wound of the right hand. This was followed by an attack of thrombophlebitis in the left lower extremity, and at the same time a gradually increasing hydrocele on the left side was first observed, probably (as was later seen) connected with venous obstruction from thrombophlebitis in the left pampiniform plexus. On admission in September, 1920 (for permission to publish this portion of the case I am indebted to the kindness of my surgical colleague, Dr. M. Schroeter) the hydrocele was of considerable size. The right testis was undescended but could be palpated as a mass about as big as a large walnut on the right side of the lower front of the abdomen. The patient was a small, rather thin, man who had formerly had gonorrhœa, but never syphilis. His Wassermann reaction (with the blood-serum) had been tried three times, once in 1919 and twice in 1920, and on all three occasions had been found negative. The urine was of rather low specific gravity and contained a trace of albumin; no sugar. Otherwise nothing special was found by ordinary examination. There was no fever; pulse, 72-96; respiration, 22-24 per minute. Brachial systolic blood-pressure (September 9, 1920): 100 mm. Hg. Blood count (September 9, 1920): Erythrocytes, 3,800,000, and white cells, 4,300 per cubic millimetre of blood; nothing special by microscopic examination. The hydrocele was operated on September 28, 1920.

About October 10, 1920, thrombophlebitis in the right internal saphenous vein was observed, and was accompanied by slight pyrexia.

\* Read at the Medical Section of the Royal Society of Medicine, London, on March 22, 1921.

Some days later on, fifteen grains of sodium citrate, three times daily, were prescribed, and a diet containing sufficient green vegetables and fruit. The sodium citrate was, I understand, continued for only three or four weeks.

On November 12, 1920, I noted that the patient was up and about in the hospital, and that there had been no fever since October 19. The superficial veins in the front of the abdomen and thorax were markedly dilated, more especially on the left side. I thought that there must be some thrombotic obstruction in the great veins of the trunk, probably in the inferior vena cava. The circulation in the dilated veins was from below upward. There was a slight tendency to œdema of the lower extremities. On November 25 I heard that there had been thrombophlebitis in one of the superficial veins on the left front of the abdomen. On November 28 there was a little temporary fever.

On December 3, 1920, the patient was transferred to my care on the medical side of the hospital. For the last three days he had passed very little urine. I thought he might have thrombotic obstruction in one or both renal veins. There was no definite ascites, but the abdomen was rather "full" (venous engorgement). On December 3 the daily quantity of urine was charted as only 200 c.c. A sample was of specific gravity, 1012, acid, free from sugar, and practically free from albumin. Brachial systolic blood-pressure (December 5): 165 mm. Hg. On December 6 I noted that the patient had had considerable pain in the lumbar-sacral region of the back during the last week, and that he had suffered much from headache during the last fortnight. Since December 3 he had apparently passed no urine at all, and it seemed clear that this anuria was due to thrombotic obstruction in both renal veins. At midday on December 6 I ordered him to have a subcutaneous injection of a sterilized solution of 2.0 grammes urea in 10 c.c. water. At 5 p. m., after a hot hip-bath, he passed about 30 c.c. of pale yellowish urine, containing a little albumin. On December 7 a solution of 4.2 grammes urea was injected subcutaneously, and at 4.30 p. m., after a hot hip-bath, he passed about 12 c.c. urine. The urinary bladder was not distended. During the night of December 7-8 he again passed about 12 c.c. urine (containing a little albumin). No further urea was given, as the patient complained that the injections

hurt him, but he was treated by hot hip-baths. About this time there was occasional vomiting. On December 9 I noted that he had passed 60 c.c. pale urine (specific gravity, 1013), containing a little albumin. Mentally he seemed quite normal and happy. On December 10 I noted that he had passed 83 c.c. of similar urine, and that the brachial systolic blood-pressure was 155 mm. Hg. It seemed that a collateral circulation through the blood-vessels in the capsules of the kidneys was being established. On December 11 I noted that he had passed 107 c.c. urine (of similar character). He had had a little vomiting, and a short epileptiform attack lasting about five minutes, with definite unconsciousness. On December 11 the hip-baths were discontinued, as patient said he felt too weak to have them. On December 12 I charted 86 c.c. urine, and on December 13 1000 c.c. (containing only a trace of albumin). The superficial veins of the collateral venous circulation on the trunk were becoming more dilated. There was dullness to percussion at the base of the right lung, apparently due to pleural effusion. Afterwards a little dullness was noted at the base of the left lung also. By December 14 the daily out-put of urine had reached 1500 c.c. On December 15 the patient was again ordered to take sodium citrate, 15 grains, three times daily. Brachial systolic blood-pressure (December 15): 140 mm. Hg. By December 16 the daily quantity of urine had reached 2000 c.c. (containing a trace of albumin). On December 17 Dr. C. Markus kindly made an ophthalmoscopic examination, and found nothing definitely wrong in either fundus. The dullness to percussion at the base of the right lung was not very great. On December 20 I noted that the superficial veins over the back of the trunk were collaterally dilated as well as those on the front.

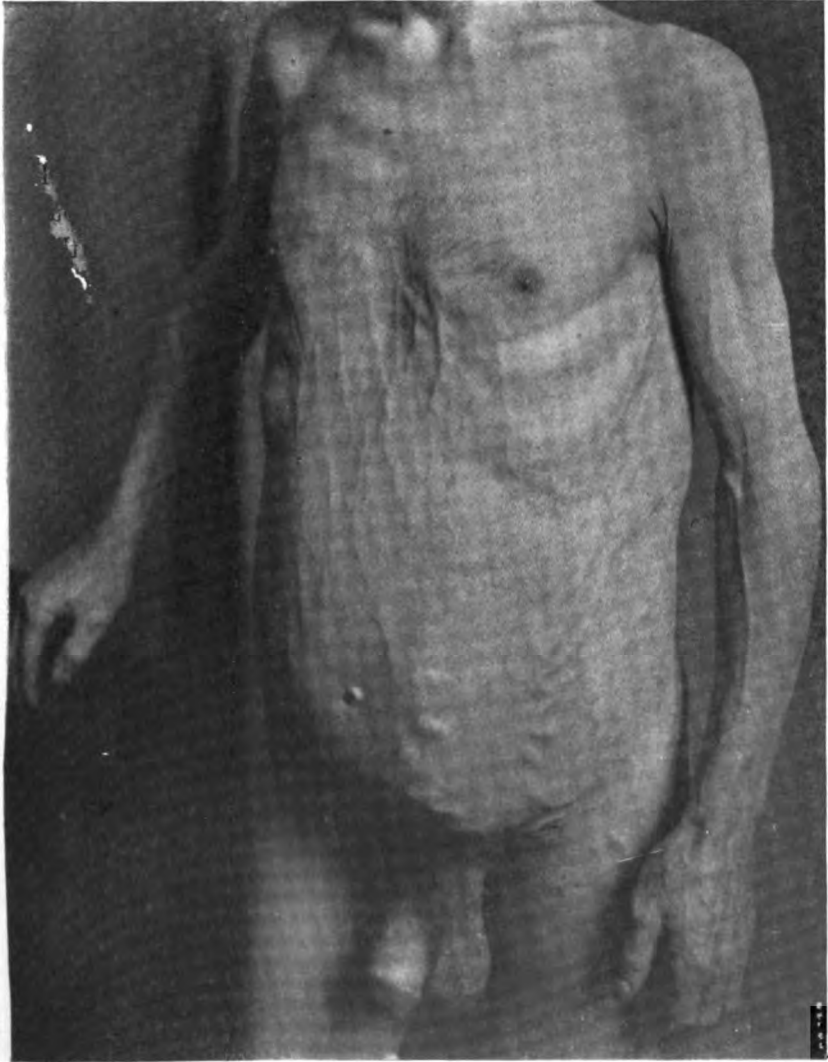
At the end of December the patient had an attack of herpes zoster of the right lower extremity.

On January 7, 1921, he was passing about 2250 c.c. urine in the 24 hours. The urine contained a trace of albumin, and microscopical examination of the centrifuge-sediment showed some granular and cellular tube-casts and white blood-cells. There had been no fever since November 28.

On January 11 the patient began to get up a little in the ward. On January 24 there was still a moderate degree of dullness at the



FIG. 1.



Thrombosis of the inferior vena cava.



base of both lungs, more marked at the base of the right lung. Brachial systolic blood-pressure (January 22): 90 mm. Hg.

Since then the patient's condition has remained about the same. There have been three transitory epileptiform attacks, two in February and one in March, evidently not of uræmic origin. He is (March 8, 1921) passing about 1450 c.c. urine in the six hours. The urine (March 23) is of specific gravity, 1015, yielding a "cloud" of albumin and containing a few granular tube-casts; no sugar. There is still slight dullness to percussion at the base of both lungs behind. The accompanying illustration (from a photograph taken on March 2, 1921) pictures fairly well the dilated superficial veins of the trunk (see Figure).

#### REMARKS

Thrombotic obstruction in the inferior vena cava and renal veins, as is well-known, occasionally occurs in cases of malignant tumor of the suprarenal glands, the kidneys or the liver. In such cases there is extension of the growth ("tumorthrombosis") through the renal vein into the inferior vena cava,<sup>1</sup> and owing to the cause of the thrombosis the termination is necessarily fatal. But it is with the cases of thrombotic obstruction of the inferior vena cava not connected with any malignant tumor that we are now concerned. The thrombophlebitis in these cases may follow traumatism, or some infection, such as typhoid fever, scarlet fever, varicella, erysipelas or, as in the above-described case, a septic wound. In such cases the patient may recover and live for a long time, but not very much is known as to the exact subsequent history and the average duration of life. Sooner or later a special liability probably develops two attacks of thrombophlebitis in the enlarged (varicose) veins. For instance, in 1907, I saw a man, aged twenty-six years, whose inferior vena cava had become thrombosed seven years previously as a sequel of typhoid fever contracted during the South African War. The thrombosis was associated with the usual œdema of the legs and compensatory enlargement of superficial veins. In six weeks'

<sup>1</sup> Cf. F. Parkes Weber, "Bilateral Hypernephroma, with Secondary Thrombosis of the Inferior Vena Cava and Terminal Uræmia," *Proc. Roy. Soc. Med., Med. Section*, 1915, viii, pp. 6-10; also F. P. Weber, "Two Cases of Primary Carcinoma in the Liver, in One of Which Thrombosis of the Inferior Vena Cava Occurred," *Proc. Roy. Soc. Med., Clinical Section*, 1917, x, pp. 30-36.

time from the first signs of the thrombosis the dilatation of the veins in the front of the abdomen had already reached its maximum. Since then he had had three attacks of thrombosis in the left lower extremity and had had "varicose" ulcers and pigmentation of the legs. This man's condition was, according to what I heard, much the same in 1912 as when I saw him in 1907.

A case of traumatic thrombosis of the inferior vena cava was shown by Dr. J. L. Bunch at the Royal Society of Medicine on June 18, 1914,<sup>2</sup> but only on account probably of a merely coincidental association with psoriasis. The patient was a man, aged thirty-three years, who three years ago, after over-exertion in running, suffered from oedema of the legs, and developed collateral enlargement of the superficial veins of the abdomen and groin.

On April 23, 1903, I examined an active, fairly muscular energetic man, aged twenty-eight and half years, for life insurance. The only point against him was that the superficial veins of the lower extremities and the lower part of the trunk were enormously enlarged, doubtless a compensatory enlargement resulting from traumatic thrombosis of the inferior vena cava. The history was that about five years previously, after a carriage accident, his lower limbs became greatly swollen and then the superficial veins of these limbs and of the lower part of the trunk began to enlarge. He was kept five months in bed altogether. Otherwise the applicant appeared in good health and free from disease, and a notable point (in regard to the extent of the thrombosis) was that his urine was free from albumin. His life was accepted for a whole life policy, with ten years extra, and on May 8, 1917, I ascertained through the kindness of Dr. A. T. Davies, that the insurance policy was still in force and that the insured man was about to pay his *last* premium.

A case analogous to the last-mentioned one—but more remarkable and instructive, inasmuch as there was ultimately a post-mortem examination, at which both renal veins were found to have been obliterated—was that of a distinguished member of our profession, who suffered from traumatic thrombosis of the inferior vena cava, when, at the age of about twenty-four years, he was house-surgeon at Addenbrooke's Hospital, Cambridge, in 1884. When I was a medical student at the university, he had shown me the

---

<sup>2</sup> J. L. Bunch, *Proc. Roy. Soc. Med., Dermat. Section*, 1914, vii, p. 257.

great enlargement of the superficial veins in his own case, and it was owing to my knowledge of his case that I ventured to recommend the life in the last-mentioned case for assurance. The whole history of this physician's case has been published by Prof. S. G. Shattock in the *Proceedings of the Royal Society of Medicine, Pathological Section* (1913, vi, p. 126), because the patient himself (the late Dr. W. Rivers Pollock), owing to the remarkable circumstances connected with the illness, bequeathed the affected parts of his body, for dissection, to the Royal College of Surgeons of England. When the thrombosis occurred (1884) he was in perfect health and won the 120-yard hurdle race for Cambridge University against Oxford, in the record time of sixteen seconds, holding his breath through the whole race. Immediately after the race was over he lay on the grass, and within a few seconds complained of pain in the lumbar spinal region. He was kept in bed for six months. The œdema of his legs and lower part of the trunk which at once supervened was followed in a few days by progressive enlargement of the superficial veins. Albuminuria, which likewise appeared at once after the injury, persisted throughout his life, a life of distinguished professional activity. Death occurred twenty-five years later (October 5, 1909), from tonsillitis and septicæmia. During the last six years of his life he had been subject to attacks of thrombophlebitis in the enlarged saphenous veins. Professor Shattock found that the inferior vena cava was converted into a flat, impervious ribbon, from the point of entry of the hepatic vein downward; both right and left renal veins were closed at their entrance into the vena cava, so that the return of blood from the kidneys must have taken place through the veins of the capsule and thence by way of the lumbar veins through the azygous vessels. Professor Shattock argues that the thrombosis was probably secondary to the rupture of the inner coat of the vena cava caused by the violent strain of the race with the holding of the breath; he compares the venous condition in question (for which he suggests the name "dissecting varix") to the arterial condition known as "dissecting aneurysm," when owing to some violent strain or previous disease a rupture of the inner coat of the aorta has occurred.

# HEART BLOCK SIMULATING PERFORATED PEPTIC ULCER \*

By I. S. RAVDIN, B.S., M.D.

AND

ELIZABETH GLENN RAVDIN, A.B., M.D.

Philadelphia

THE interest in the cases reported in this paper lies in the fact that both were diagnosed as "acute surgical catastrophies" of abdominal origin, when in their final analysis they proved to be medical conditions, due primarily to extra-abdominal pathology. Both were admitted to surgical services with a diagnosis of perforated gastric or duodenal ulcer.

It is a well-known observation that pain is frequently reflected from the site of the lesion causing it. This is especially true of pain from lesions in the chest. Nevertheless we often disregard the fact that abdominal pain may be caused by a medical condition, the etiology of which may be intra or extra-abdominal. The literature records the reports of many operations on patients for supposed surgical abdominal pathology, when the patient was really suffering from pneumonia, diaphragmatic pleurisy, tabes dorsalis, and typhoid fever. Nuzum reported 1000 cases of tabes dorsalis, 8.7 per cent. of whom were subjected to surgical operations for gastric or duodenal ulcer, gall-bladder disease, appendicitis or other surgical lesions.

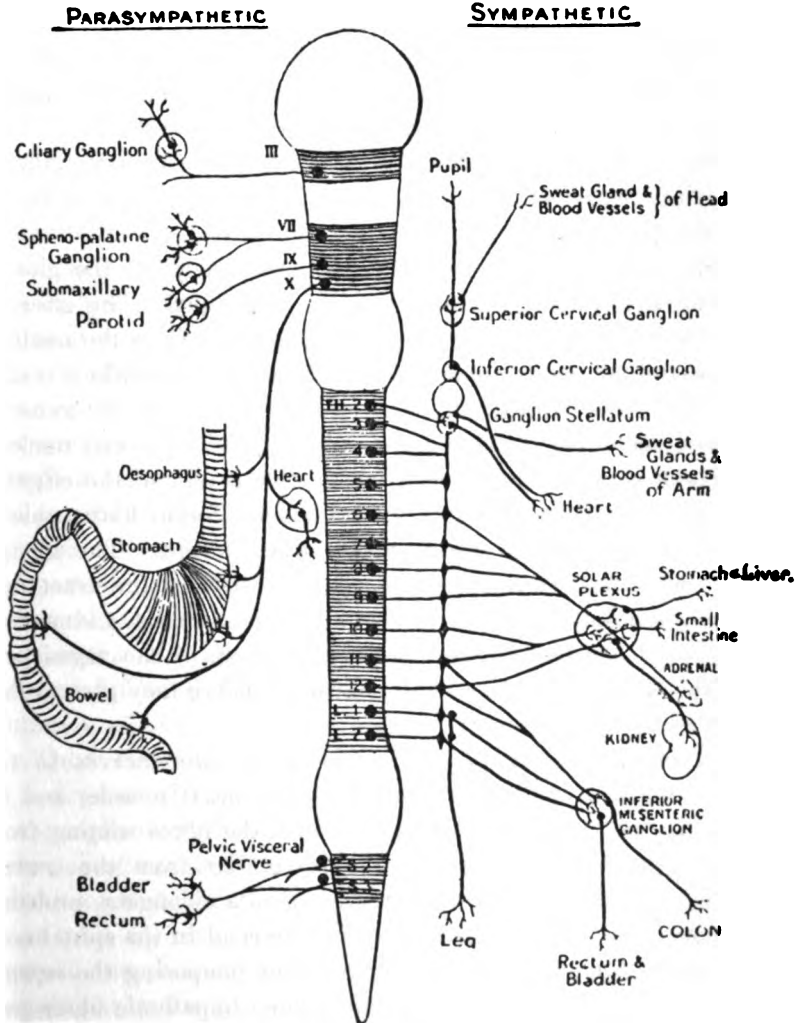
Many clinicians have emphasized the extensive distribution of cardiac pain, particularly of anginal pain, without, however, explaining various atypical sites of this pain on an anatomical basis. Abdominal discomfort is a not uncommon symptom in almost any cardiac lesion; however, it is not frequently met with in the form of epigastric crises. The cases reported here are of patients suffering from heartblock, one having the Adams-Stokes Syndrome. Both of these patients were sent in by their physicians for immediate operations, a diagnosis of perforated ulcer having been made.

---

\* From the Hospitals of the University of Pennsylvania.

Pain in cardiac conditions must be explained on an anatomical and a physiological basis. The accompanying explanation and

FIG. 1.



Scheme of the general arrangement of the Autonomic Nervous System, the distribution of the Sympathetic and Parasympathetic portions being contrasted on the two sides of the diagram. (Gaskell).

description of nervous innervation is merely an attempt on our part to clarify the symptoms of our cases. Disturbed action of the auto-

autonomic nervous system as the result of lesions of viscera is the chief cause of the symptoms of visceral disease. In this paper we shall refer to the autonomic nervous system as the combined sympathetic and para-sympathetic (or cerebral autonomic) systems. Many of these symptoms are reflex in character. In order to clearly interpret them we must understand the functions of the nerves supplying the viscera together with the intricate inter-relation of these nerves in the autonomic system and with the cerebrospinal system.

The nerve supply of the heart is from both, the para-sympathetic and the sympathetic systems, the para-sympathetic innervation being through the vagus nerve. (Fig. 1.)

Centrally the vagus is connected with the nuclei of the glosso-pharyngeal, the spinal accessory, and the trigeminal. The efferent fibres of the vagus and the glosso-pharyngeal arise from the nucleus ambiguus and the nucleus dorsalis. The former nucleus which is also the point of origin of the cerebral portion of the ramus internus of the spinal accessory is a somatic motor nucleus. The dorsal nucleus is mixed and contains motor cells which give origin to the efferent para-sympathetic fibres and cells around which afferent fibres, chiefly from the vagus, break up into terminal arborizations. The tractus solitarius with its accompanying nucleus constitutes the tract and nucleus of termination of the glosso-pharyngeus, vagus, and nervus intermedius of the facial. As the afferent fibres of the vagus pass through the tractus spinalis of the trigeminal nerve they give to this nucleus their somatic sensory fibres.

The sympathetic, which carries the accelerator nerves to the heart, is connected on the one hand with the heart muscle, and on the other, in the sympathetic ganglion, with the fibres coming from the central nervous system. The fibres coming from the central nervous system have their origin in the medulla oblongata, probably in the floor of the fourth ventricle. They descend in the spinal cord and emerge in company with the nerve fibres composing the second, third, fourth, and fifth thoracic nerves. The sympathetic fibres pass into the sympathetic chain through the white rami communicantes, then to the stellate ganglion, the ganglion on the annulus of Vieussens, and to the inferior cervical ganglion around the nerve cells of which the terminal fibres make their pre- and post-ganglionic connection. It is from the nerve cells of the stellate ganglion, the gang-



lion on the annulus of Vieussens, and the inferior cervical ganglion that the post-ganglionic sympathetic accelerator fibres to the heart arise.

The connection of the cardiac plexuses to the thoracic segments through the sympathetic nerves is by the white rami communicantes, afferent as well as efferent impulses being carried. The cervical sympathetic ganglia are connected to the spinal nerves by gray rami communicantes only, the white rami not being present until after the second thoracic segment, or at best, the first is reached. As Gaskell has so clearly pointed out, all the connector neurons, which form the white rami communicantes running from the central to the sympathetic nervous system, leave the cord from the second dorsal to the second lumbar segments. Through the upward prolongations of the white rami communicantes of the upper thoracic segments the cervical sympathetic ganglia have a connection with the spinal nervous system. The connector pre-ganglionic fibres of the sympathetic do not necessarily connect with the excitors at the same level of the ganglionic chain. The medullated pre-ganglionic fibre may end in the ganglion with which the ramus is connected, or it may course upward or downward to another ganglion, or it may pass through the sympathetic trunk to end in a peripheral ganglion, that is, a lateral, collateral, or terminal ganglion. It is through this intricate connection that a single segment may be connected with a number of cells, at different levels, and it is this which accounts for an inter-radiation of sympathetic impulses.

There is according to Gaskell and Langdon Brown, no evidence of a division of the afferent fibres in the sympathetic into pre- and post-ganglionic portions, nor is there satisfactory evidence of the presence of non-medullated afferent fibres. The afferent fibres have trophic or nutrient centres in the posterior root ganglion, and from these pass by way of the white rami communicantes through the sympathetic chain. Gaskell says, "seeing then that all the fibres entering into the posterior root ganglia are medullated, it follows that all non-medullated fibres are efferent, non-afferent, and that the so-called sympathetic system is not a complete central nervous system but consists purely of excitor neurons."

The white rami or connector neurons of the second to the fifth thoracic segments inclusive are directed chiefly upward to be distri-

buted to the cardiac plexuses through the inferior cervical ganglion, the ganglion on the annulus of Vieussens, and the stellate ganglion. The thoracico-lumbar outflow from the sixth dorsal to the third lumbar segments is directed chiefly to the inferior part of the sympathetic trunk and its divisions, and thence distributed to the abdominal viscera. A few of these fibres supply certain of the thoracic viscera, *i.e.*, lungs, aorta, and œsophagus, directly. The lower thoracic ganglia form plexuses on the thoracic aorta and by means of the splanchnic nerves are the chief source of the celiac plexus. The nerves of the liver, which are chiefly of the non-medullated type, are derived from the left vagus and the celiac plexus of the sympathetic. The sympathetic supply to the liver is through the white rami of the sixth, seventh, eighth and ninth thoracic nerves.

Langdon Brown says that "in the thoracic and abdominal viscera most of the afferent fibres which on electrical stimulation give rise to pain, pass by the sympathetic and not by the vagus." Langley has summarized the special features of the afferent nerves to the viscera as follows: "The healthy viscera give rise to little or no sensation when cut. This is probably due to the comparatively small number of sensory fibres running in a given area, the total number distributed to the whole of the viscera of the cat being about the same as the number present in a single posterior spinal root. In pathological conditions, however, cutting may be painful, and strong contraction may give rise to intense pain. The localization of pain is very imperfect. In pathological conditions the viscera readily give rise to pain and tenderness in the body wall." Luciani says, "The deep organs innervated by the sympathetic normally feel little pain but they have a very high latent sensibility which may become apparent under abnormal conditions, particularly in inflammation." Hurst (Hertz), in commenting on the work of Lennander and MacKenzie regarding the improbability of true visceral pain, reminds us that a nerve ending may be sensitive to one form of stimulation, the adequate stimulus, but insensitive to all others. This adequate stimulus he believes to be tension.

Both Foster and Sherrington have called attention to the improbability of the presence of pain fibres in the sympathetic nerves, when they may never be used during the lifetime of an individual. Hurst finds it difficult to explain how a special mechanism of this

kind could have originated, or, once originated, could be preserved through several generations, when it was seldom, if ever, used.

The visceral afferent fibres found in the sympathetic through their connection in the posterior root ganglion, terminate in definite segments of the cord. This arrangement is such that an organ when diseased, expresses its reflexes in a regular manner. It is through such an arrangement that diseased viscera show reflex sensory or motor activity. This paper deals chiefly with the subject of reflex sensory activity which is closely associated with referred pain. Hilton, in 1863, first discussed the probability of cutaneous pain being the result of a visceral lesion, the reflex taking place in a central connection. In the following year, Lange published a paper which was a precursor of the work of Head, Ross, and MacKenzie, but which was overlooked because it was written in Danish. He described an afferent impulse from a viscus carried to the cord and referred out in a spinal nerve originating in the same segment which was interpreted by the brain as originating in the termination of that nerve.

MacKenzie developed a unique theory of referred visceral pain. Normally the impulses, coming to a segment from the viscera through the afferent nerves, do not make a conscious impression on the individual. When, however, as the result of a pathological lesion in a viscus, the increased stimulation renders hyperirritable that segment or segments of the cord, all the nerves arising from that hyperirritable segment are much more easily stimulated. This hyperirritability is transitory and may be due to spasmodic bombardment of the segment by stimuli from the diseased viscus. The result is that normal somatic afferent impulses reaching the hyperirritable segment give rise to painful impressions which are referred to the peripheral tissues, since sensory nerves when stimulated in any part of their course, refer the resulting sensation to the peripheral end organ. MacKenzie demonstrated this in many cases of visceral disease by the hyperalgesic and hyperæsthetic state of similar segmental portions of the external body wall and by exaggerated motor reflexes. The studies of Head concerning the areas of cutaneous hyperalgesia occurring in visceral disease confirmed the work of MacKenzie in that he showed that the hyperalgesic areas are identical with the areas which receive their sensory nerve fibres from the spinal seg-

ments to which the afferent fibres from the diseased viscera pass. Thus it is seen that diseased viscera make us conscious of their lesion by referred pain in the terminations of the spinal nerves or in the trigeminal nerve.

Ross, Head, Kast, Maltzer, and Hurst among others have advocated the theory of true visceral pain of one kind or another. The recent work of Leriche, on sympathectomy in causalgia, is used by some as a confirmation of this theory. However, none of the work has been conclusive enough to demonstrate true sympathetic pain fibres. We believe that visceral pain is brought about by the increased afferent sympathetic impulses producing, as MacKenzie has said, a hyperirritable segment of the cord. A normal stimulus passing along the somatic sensory fibre, because of the over-excitability of the segment, is sufficient to produce pain. This afferent impulse being conducted to the perception centres of the brain is referred by it to the end organ of the somatic sensory nerves of that particular segment.

In order to fully appreciate the viscerosensory reflex we must review the distribution of the nerves which play a part in the syndrome. We owe much of our knowledge of segmental distribution to the work of Head. The cervical nerves (second, third and fourth) supply the skin of the back of the head, neck and the shoulders. The fourth cervical also descends to the front of the chest as far as the third rib. Here it overlaps the distribution of the second thoracic. The anterior rami of the fifth cervical to the eighth cervical inclusive with the aid of the first and a branch from the second thoracic form the brachial plexus.

The skin of the anterior and lateral walls of the chest is supplied by the second, third, fourth, fifth and sixth thoracic, the anterior ramus of the sixth thoracic supplying the skin in the epigastric triangle. The intercosto-brachial nerve, which has its origin in the second and third thoracic nerves, supplies the skin of the upper and inner side of the axilla and arm. It is through the connection of the intercosto-brachial and the greater and lesser internal cutaneous nerves (from the eighth cervical and first thoracic, and the first thoracic respectively) that reflexes from the visceral lesions in the chest radiate down the inner side of the upper extremity.

*Discussion.*—The frequency of digestive disturbances in myocardial disease is well-known. Vague epigastric pain is also a fre-

quent symptom in cardiac lesions. Riesman has noted the frequency with which grave myocardial weakness may masquerade as dyspepsia. He has emphasized the point which has been noted by many other clinicians, that is, the regularity with which cardiac pain comes on after exertion, especially if the exertion follows a meal. However, he states that Adam-Stokes disease is commonly painless and makes no mention of a condition such as we are reporting. Stengel related to the authors a case of mitral stenosis with pain so closely resembling gall-stone colic that a laparotomy was done.

Osler, Krumbhaar, Bachman, Ashton, Norris and Lavenson, Hays, and Carter have reported cases of heart-block with varying degrees of upper abdominal symptomatology. But none of the cases in the literature is similar to ours.

In endeavoring to account for the severe pain in our case, two explanations can be offered. The first is the one commonly offered, that is, referred pain from the heart. It is possible that afferent impulses from the heart may be carried by the vagus or by the sympathetic. We do not believe that it is possible to explain this pain by vagus innervation. It is true that pain of a reflected type due to vagus stimulation is met with in cardiac disease, this pain, however, is felt in the face and is explained by the very close connection between the trigeminal and vagus nuclei in the medulla. Gaskell explains this by expressing the opinion that the vagus contains visceral afferent branches of a primitive series of nerves, whose somatic sensory roots are in the sensory portion of the fifth nerve. There is no direct connection between the vagus and the spinal nerves which could account for reflected abdominal pain. It is a well-known clinical observation that in operations in the abdomen with the use of spinal anaesthesia, cholecystectomy can be performed without pain. No pain is experienced when the bowel is handled. The vagus, having its origin much higher than the phrenic, could not be affected by the anaesthetic before respiratory symptoms were experienced. If the vagus itself contained pain fibres, the upper abdominal operations could not be painlessly done under spinal anaesthesia.

Gaskell, Pottenger and Langdon Brown agree that the spasm of the pyloric sphincter is undoubtedly due to sympathetic innervation and that the vagus supplies inhibitory fibres to this muscle. Howell,

Brubaker and a number of other physiologists state that the vagus supplies the excitator motor fibres to the pylorus, and the inhibitory fibres to this sphincter are from the sympathetic system. Gaskell and Langdon Brown assert that this is probably the reverse of the actual innervation. It is therefore impossible for a cardiac lesion to cause pylorospasm through vagus stimulation. Therefore, when epigastric pain is attributed to pylorospasm secondary to cardiac lesions, the sympathetic mechanism only should be considered and the difference in segmental innervation of the heart and the pyloric sphincter through the sympathetic makes this an improbable explanation.

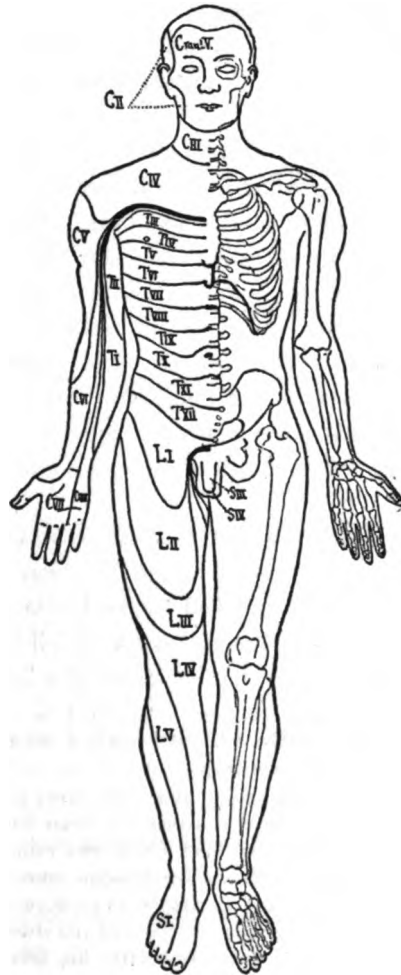
The sympathetic supply to the heart is chiefly from the second, third and fourth thoracic segments although the fifth assists in this innervation. The sensory roots of the second, third, fourth and fifth thoracic nerves supply the anterior chest wall. The first and second thoracic assist in the innervation of the axillary and ulnar side of of the upper extremity. The left third and fourth thoracic are chiefly concerned in supplying the cutaneous region of the precordium. The sixth thoracic supplies the skin of the epigastrium. None of the fibres to the heart emerge from the sixth thoracic segment. In referring to the diagram on the root fields (Fig. 2) it must be remembered that the radicular areas extend on either side of the line bearing the corresponding segment numbers, so that while each segment is supplied by its corresponding spinal nerve, the nerve above and one below also, assist in innervating it.

The liver receives its sympathetic innervation through the white rami from the sixth to the ninth segments inclusive and the left vagus, the sixth and seventh forming the chief innervation. The capsule of the liver also receives filaments from the phrenic nerve, coming from the third, fourth and fifth cervical roots.

If the pain in our case is to be explained by a cardiac viscerosensory reflex we should expect in the first place to find the pain chiefly on the left side and in the distribution of the second, third and fourth thoracic nerves. Secondly, in accordance with the work of Head and MacKenzie, we should expect areas of cutaneous hyperalgesia over the same somatic sensory distribution. We might even expect pain over the right side of the chest in the same segmental distribution. Adjacent segments may be rendered hyperirritable also, but the maximum excitability is in those segments directly involved.

Neither of our cases during the attack had acute pain or hyperalgesia in the distribution of the upper thoracic segments. MacKenzie has called attention to the pain in the upper abdomen in cases

FIG. 2.



Cutaneous sensory distribution of spinal nerves.

of rapid cardiac decompensation with consequent rapid dilatation of the liver and he says, "The hyperalgesia of the tissues covering the liver is due to stimulation of the sensory centres in the spinal cord by the afferent sympathetic fibres from the engorged liver." He has found the hyperalgesia to extend over a larger area than the

liver itself. The stimulation of the afferent fibres from the liver is due to a venous engorgement of that organ which puts the peritoneal coverings of the liver together with Glisson's capsule under increased tension. This offers the second explanation of upper abdominal pain in cardiac lesions.

Another evidence of the visceral congestion in the cases of heart-block is the frequency of gastro-intestinal disturbances and vomiting of blood.

Carter, Olser, Hay and Barringer have reported instances of heart-block in which the patients had enlarged livers, and complained of varying degrees of upper abdominal pain. In none of the cases, however, was the pain severe enough to mask the primary diagnosis. Tension of the liver due to venous stasis will cause viscerosensory and visceromotor reflexes through the sixth, seventh, eighth and ninth thoracic segments, the somatic area thus chiefly involved extending from the level of the epigastric notch to just above the umbilicus. The visceromotor reflex is never as definitely localized as the viscerosensory reflex. Both of our cases had a marked enlargement of the liver.

As will be seen from the histories, there was tenderness in the upper right quadrant with rigidity, and this was the area of maximum pain. There was also marked hyperalgesia.

There is no definite experimental work which has proven without doubt the etiology of this pain but we favor the latter explanation.

CASE I.—G.H.S., male, aged forty-one. Admitted to the Surgical Wards of the University Hospital, November 22, 1919, with a chief complaint of severe pain in the upper abdomen and vomiting.

H.P.I. In October the patient had an attack of severe pain in his epigastrium. During the next few days he had several attacks of lesser degree. He occasionally had pain in relation to the taking of food which was relieved by vomiting. No blood was ever noticed in the vomitus. The attacks were sometimes relieved by belching. In November, 1918, he had an attack of syncope and he was taken to a Philadelphia hospital where a diagnosis of "ulcer of the stomach" was made. The present attack began with a little discomfort after his breakfast, but he went to work as usual. He felt more uncomfortable and was nauseated after his dinner. After his supper he vomited. The vomitus consisted of stomach contents. After a short period of belching he again vomited, and this time he said he vomited blood. He was free from pain until early the next morning when he was awakened by violent epigastric pain and vomiting. His doctor, who was called immediately, stated that his temperature was subnormal, his pulse about 130, that he was vomiting blood, and that he had marked muscular rigidity and tenderness in the upper right abdomen.



P.M.H. Diseases of childhood. Chancre in 1902. Bubo on the right side in 1903. Gonorrhoea in 1913.

F.H. Father died of cancer of the liver. Mother died of cancer of the stomach. One brother has the same trouble as the patient.

S.H. Married. Wife well; no pregnancies. Patient is a chemical worker. Fair hygiene. Smokes cigarettes to excess. Drank excessively until two years ago.

*Physical Examination.*—Adult male about forty years old. Good bony frame-work and musculature. He was having very severe pain which was chiefly in the epigastrium. He was slightly cyanotic, cold, clammy, with a rapid running pulse. Blood-pressure, systolic 110, diastolic 80.

*Chest.*—The percussion note is impaired over the whole right lung. Fine and coarse râles can be heard over the right lung especially low in the axilla and posteriorly. The breath sounds are normal throughout.

*Heart.*—A slight apex pulsation is seen in the fifth interspace in the nipple line. It is feeble.

The supra-cardiac dullness is 6 cm., R.B. cm.; L.B. 13½ cm.; R.O., 14 cm.; L.O., 17 cm.; height, 15 cm.

The heart sounds are of poor muscular quality and a snappy first sound is heard. No murmurs are audible. There is reduplication of the second sound at the base of the heart. The rate was 132 and there is an extra-systolic arrhythmia.

*Abdomen.*—A little full but not distended. There is considerable tenderness over the upper half of the abdomen quite marked at either side of the mid-line but most marked at a point in the mid-line about half way between the umbilicus and the ensiform. He had some muscular rigidity but it was not board-like. There was hyperperistalsis. The liver dullness extends from the fifth rib to a considerable distance below the rib margin, but the edge cannot be felt because of the extreme discomfort caused by palpation. The abdomen was too tender to palpate the spleen.

The extremities are negative.

Just after the patient was admitted to the ward there occurred an attack of syncope, with convulsive movements of the left arm, and head and complete absence of the radial pulse. No cardiac sounds were audible. Dr. Alfred Stengel observed this attack and recognized it as an Adams-Stokes Syndrome with complete heart-block. The pulse re-

turned at a rate of thirty-two per minute, while the jugular pulsations were about 120. The blood pressure was 50-25. He vomited several times, one time a small amount of blood. Six hours afterward, the pulse was 48. He had several convulsions after the first one.

Twelve hours later the pulse was 120. The next morning the patient vomited coffee ground material so a diagnosis of probable gastric ulcer and a luetic affection of the bundle of His was made.

The leucocytosis on admission was 15,000 and the following day it was 28,000. The polymorphonuclears were 90 per cent. on admission.

Wassermann was delayed negative. The urine showed a heavy cloud of albumin and many hyaline and dark granular casts. Blood urea was 45 mm. per 100 c.c.

No electrocardiographic tracings were taken during an attack and therefore, later showed only a ventricular extra-systole.

The patient showed a gradual improvement during the next nine days. On the tenth day the patient was given a test meal preparatory to a gastro-intestinal X-ray. He took a small duodenal tube without any difficulty. About 2 ounces of stomach contents were removed when the patient was asked to lie on his left side. Upon turning he had a sudden attack with convulsive movements of his head and neck. There was horizontal nystagmus; his breathing became labored and shallow, and then ceased. No sounds could be heard over the heart, and no radial pulse could be felt. Artificial respiration was of no avail.

The necessity of a careful history is well-illustrated in this case. Since the predominant symptoms at the time of admission seemed to point to a surgical condition the emphasis in the history was placed on the abdominal condition. A more complete history taken at leisure brought out the following facts: The patient for over a year had noticed attacks of dull precordial pain on exertion which pain radiated down the left arm and was relieved by rest. Coincident with the epigastric attacks he had precordial pain much less severe than the abdominal pain with radiation to the arm at times extending to the fingers. One year ago he had fainted while walking on the street, but he had no pain at that time. He occasionally had palpitation.

## AUTOPSY PROTOCOL (DR. LUCKE)

Gross anatomical diagnosis:

Histological diagnosis:

*Aorta—*

Toxic intimal athero-sclerosis

Toxic athero-sclerosis

*Heart—*

Hypertrophy; chronic interstitial myocarditis; chronic and partly organizing thrombus in left ventricle; chronic sclerotic mural endocarditis; arterio-sclerosis; calcification of coronary arteries; probably obstruction of anterior coronary artery. Gumma of the heart

Chronic interstitial myocarditis; adherent partly organizing thrombus; sclerosis of coronary arteries. Gumma of the bundle of His

*Lungs—*

Congestion and oedema; bilateral hydrothorax

Chronic passive congestion

*Duodenum—*

Chronic passive congestion

*Liver—*

Chronic passive congestion

Chronic passive congestion

*General Statements.*—Body is that of a white male, about forty-five years old, weighing about 90 k., and measuring 175 cm.; robust build, well-developed musculature; well-nourished; skin smooth; no scars or oedema; no glandular swelling. Rigor mortis not present; marked livor mortis present in dependent parts.

## INTERNAL INSPECTION

*Abdomen.*—Abdominal fat is about 2 cm. in thickness; moist and dark yellow. Abdominal muscles are dark fleshy-color and fleshy consistency throughout. Peritoneum is smooth, moist and glistening. Few drops of clear, serous fluid in pelvis. Omentum and mesentery are very fatty. Diaphragm on right extends to fifth rib, on left side to fifth interspace. Abdominal organs are in normal position.

*Heart.*—Pericardial sac free from fluid. Pericardium is moist; over right wall, contains about 5 mm. of fat. Over left heart, a fine grayish membrane firmly adheres to the surface, giving it a dull appearance. Near the apex there is a firm, fibrous adhesion between

the two layers of the epicardium. Heart is very large, measuring 14 cm. in its greatest length, and 12 cm. in its greatest width. Heart muscle is very firm throughout the entire wall, but especially near the apex. Near the apex the muscle is thin, averaging 8 mm., while further up it averages 20 mm. in thickness. This latter measurement applies to the left ventricular wall. The right ventricular wall averages 9 mm. In the left ventricular cavity there is a firm laminated thrombus adherent to the heart muscle in lower third of the ventricular cavity and reaches into the cavity by pedunculated mass about the size of a marble. At the junction of the clot and heart muscle, lamination grayish and brown may be seen. Endocardium is thickened and in places quite opaque. Cordæ tendinæ are thin and separate. Papillary muscles possess white fibrous tops. Valve leaflets are thin and delicate. Aorta measures 8; mitral, 10; tricuspid, 13, and pulmonary, 8½ cm. Coronary arteries are stiffened; anterior branch is much calcified, and possesses so narrow a lumen that section is not possible. The intra-ventricular septum is brownish in tint; throughout it one sees fibrous scars. A small gummatous lesion involves the auriculo-ventricular bundle. Weight, 650 grams.

*Esophagus, Stomach, Duodenum.*—Esophagus is of normal diameter; mucosa is grayish. Submucosal veins are nowhere prominent. Stomach is of average size; walls of normal thickness; mucosa normally rugous, and overlaid with normal amount of slimy mucus; There are nowhere any submucous hemorrhages nor are submucous veins prominent. Continuity of mucosa is everywhere unbroken. Duodenum shows very prominent submucous veins. Here and there submucous hemorrhages may be seen; no break in continuity of mucosa could be discovered.

*Liver, Gall-bladder.*—Liver weighs 2100 gm., measures 30 x 21 x 10 cm. Lower margin is slightly rounded; consistency is firm; capsule is smooth and translucent; surface lobulation is prominent. Cut surface is moist and bloody; lobulation is distinct; centres of lobules, deep red. Gall-bladder is normal in size; contains thick, tarry bile; mucosa is honey-combed.

*Pancreas.*—Is of normal size and consistency; is light dusky-brown in color, except at the periphery where it is of more yellowish color.

*Mesentery, Intestines.*—Mesentery is very fatty; mesenteric lymph-nodes are of normal size; grayish in color. Intestines—serosa is smooth; mucosa is coated with mucus.

The histological examinations confirmed the gross findings.

**CASE II.**—H.M., male, aged fifty-three. Admitted to the Polyclinic Hospital, surgical service of Dr. G. P. Muller, with a chief complaint of severe upper abdominal pain.

**H.P.I.** The patient was feeling perfectly well until one half an hour after his dinner when he was seized with sudden, severe, cramp-like pains in his epigastrium. The pain was severe enough to make him feel faint. The pain was over the entire upper abdomen and did not radiate except slightly to the right iliac fossa. He has been nauseated but has not vomited although he has tried to a number of times. His bowels have moved several times today and he has expelled flatus. The patient states that he had a similar attack one year ago and another one month ago, neither of which was as severe as the present one. After the first attack he thinks he was slightly jaundiced. He has had some vague discomfort in the epigastrium after meals with occasionally actual pain. He frequently has sour eructations but he never has vomited.

**P.M.H.** No diseases except measles and whooping cough in infancy. No operations. He denies any venereal history.

**F.H.** Negative.

**S.H.** Patient is a night watchman. He smokes to excess. Some years ago he was a heavy drinker.

*Physical Examination.*—The patient is a very large adult white male about fifty-three years old. He lies with his lower extremities flexed on his abdomen evidently suffering severe pain. His skin is cold and clammy. His temperature is subnormal, and he is somewhat cyanosed. There is no icterus. There is a very rapid venous pulsation in the neck.

*Chest.*—The chest is hyperresonant throughout, and moist râles were heard in both the lungs. The expansion is somewhat limited.

*Heart.*—The apex beat is in the sixth interspace and just to the left of the mid-clavicular line. The heart sounds are weak, but no murmurs are heard. The heart rate is 42 and irregular.

*Abdomen.*—There is marked rigidity over the whole upper abdomen but this is not board-like in quality. The upper abdomen is very tender especially in the epigastrium. The liver is palpable below the costal margin and there is tenderness in this region. Active peristalsis is heard. There is no dullness in the flanks. No distention.

Extremities, negative; blood-pressure, systolic 95, diastolic 60; leucocytes, 16,000.

We at once requested that the patient be seen by the medical side of the hospital. They agreed that the condition was that of heart-block. It is to be regretted that the hospital does not have a string-galvanometer.

After four days in the hospital the pain had disappeared, and the patient was released at his own request with a heart rate still around forty.

Gastric analysis, normal; Wassermann, negative; urine, negative.

It is of course possible that this case may have had an intra-abdominal lesion but the negative laboratory reports and response to rest in bed makes it probable that the condition is similar to the first case reported.

#### BIBLIOGRAPHY

- <sup>1</sup> ASHTON, T. G.; NORRIS, G. W.; LAVENSON, R. S.: *Amer. J. Med. Sc.*, 1907, cxxxiii, 28.
- <sup>2</sup> BACHMAN, G.: *Amer. J. Med. Sc.*, 1909, cxxxvii, 342.
- <sup>3</sup> BARRINGER, T. B.: *Arch. Int. Med.*, 1909, iv, 186; Publications of Cornell Univ. Med. School, 1909, ii.
- <sup>4</sup> BROWN, LANGDON W.: "The Sympathetic Nervous System in Disease," London, 1920.
- <sup>5</sup> CARTER, E. P.: *Arch. Int. Med.*, 1914, xiii, 803.
- <sup>6</sup> CLAYTON, T. A.: *Amer. J. Med. Sc.*, 1912, cxliv, 697.
- <sup>7</sup> EUSTERMANN, G.: Collected Papers of St. Mary's Hospital, 1911, ii, 193.
- <sup>8</sup> FOSTER, M.: "Text-book of Physiology," London, 1891, 4th ed.
- <sup>9</sup> GASKELL, W. H.: "The Involuntary Nervous System," London, 1916.
- <sup>10</sup> GIBSON, G. A.: *Brain*, London, 1905, xxviii, 52.
- <sup>11</sup> GOODALL, J. S.: *Lancet*, 1920, cxviii, 909.
- <sup>12</sup> HEAD, H.: *Brain*, 1893, xvi, 2; 1894, xvii, 339.
- <sup>13</sup> HERTZ (HURST), A. F.: "The Sensibility of the Alimentary Canal," London, 1911.
- <sup>14</sup> HILTON, J.: "Lectures on Rest and Pain," London, 1863.
- <sup>15</sup> KAST, L., AND MELTZER, S. J.: *Mitteil. a. d. Grenzgeb. d. Med. u. Chir.*, 1909, xix, 586.
- <sup>16</sup> KRUMBHAR, E. B.: *Univ. Penn. Med. Bull.*, 1908-09, xxi, 278.
- <sup>17</sup> LANGE: Quoted by Hertz.
- <sup>18</sup> LANGLEY, J. U.: *Brain*, 1903, xxvi, 23.
- <sup>19</sup> LUCIANI: Quoted by Pottenger.
- <sup>20</sup> LENNANDER, K. G.: *Centralbl. f. Chir.*, 1901, xxviii, 209; *Jour. Amer. Med. Assn.*, 1907, xlix, 836.

- " MACKENZIE, SIR J.: "Diseases of the Heart," *Oxford Med.*, London, ii, part 1; "Symptoms and their Interpretation," 3rd ed., 1918; "Diseases of the Heart," London, 1918, 3rd edition.
- " NUZUM, T.: *Jour. Amer. Asso.*, 1916, lxvi, 482.
- " OSLEE, SIR W.: *Lancet*, 1903, ii, 9.
- " POTTENGER, F. M.: "Symptoms of Visceral Disease," St. Louis, 1919.
- " RIESMAN, D.: *Penn. Med. Jour.*, 1916, xix, 343.
- " ROSS, J.: *Brain*, 1888, x, 350.
- " SCHMIDT, R.: "Pain," 2nd ed. (Trans. by Vogel and Zinsser), Phila., 1911.
- " SHERRINGTON, C. S.: In Schäfer's *Physiology*, 1900, ii, 990.
- " STENGEL, A.: Personal communication.

## NEUROSES AND PSYCHONEUROSES AND THE THERAPEUTIC VALUE OF FOOD

By JAMES J. WALSH, M.D., Ph.D.

Medical Director of Fordham University School of Sociology, Professor of  
Physiological Psychology at Cathedral College, New York City, N. Y.

---

DURING the course of the past fifteen or twenty years I have seen a good many patients suffering from rather severe, or at least what to them were very disturbing, neurotic and psychoneurotic symptoms, for whom practically the only treatment that proved to be needed was such definite and insistent prescription with regard to the taking of food as would bring about a distinct improvement in nutrition. The basis of their complaints was lack of proper nutrition, and the principal therapeutic indication in their cases was just a good gain in weight. So many of these patients have come to me after having been treated by other physicians—as a rule each of them giving the story of having been under the care of at least two or three physicians, who had tried all sorts of remedies on them, commonly without much success or only temporary improvement—that I have come to wonder whether the therapeutic value of food itself is as much appreciated as it should be.

Dietetic treatment has always been considered of very great value by physicians all down the centuries, but in a great many minds dietetics seems to mean limitation of food or the elimination of dietary elements that are supposed to disagree. It must not be forgotten that it should signify also sufficiency both in quantity and variety of what is eaten by the patient, in order to keep him in as good physical condition as possible. Nervous patients, it must not be forgotten, in their solicitude about themselves are likely to conclude that various articles of food disagree with them. Not a few of them acquire prejudices particularly with regard to the most nutritious articles of food—milk and eggs and perhaps butter and the fats. Often they have been encouraged in this finickiness by physicians who still retain the old-fashioned idea of dyspepsia as a disease in itself.



The patients who have been benefited by simple gain in weight have been sufferers from nervous symptoms of many kinds. Practically all of them have had exaggerated knee jerks and at least increased ankle jerks or Achilles' reflexes; at times they have had some disturbance of station and a definite tremor when the hands were held out, and a large number of them complained of periodical headaches with some disturbance of sleep and a tendency to over-react to sudden noises or surprises of any kind beyond the ordinary. In a word they presented the familiar picture of the nervous patient often so difficult to treat successfully and always prone to have a chronic condition, finding his way from one physician to another, and very often ending up in the hands of some quack who instead of dosing him with medicine does something to him that creates an impression on his mind that he is going to get better, encourages him to eat, brings about a gain in weight. Then he becomes a booster for some particular form of charlatanism; another convert to one of the many absurdities of therapeutics, so rampant in our time, which illustrate so well how little of reasoning there is in our generation.

So many of these patients have been benefited simply by an increase in the amount and variety of food taken, until they make a considerable gain in weight, that at last I have come to feel that every thin neurotic ought to be definitely treated in the first instance with the idea of seeing that he shall gain more weight, and only after that very practical bit of therapeutics has been accomplished should the question of any specific treatment that may be needed for him be taken up.

Not a few of the patients that I have seen who were benefited simply by an increase in nutrition complained of annoying psychoneurotic symptoms. Practically all of them had digestive complaints—sour stomach, heart burn, eructations of gas and discomfort from its collection—symptoms which are much more often due, when there is no gross gastric lesion present, to undereating than overeating. A stomach that has not enough to do gives its possessor a good deal of trouble. This reacts upon the mind and a vicious circle of influences is formed until a state resembling hypochondria often develops. This is often much more psychoneurotic than neurotic indigestion, though most of the patients have come with the

ready-made diagnosis of neurotic indigestion or dyspepsia or the longer phrase, which so many of them like, neurotic gastro-intestinal indigestion. They suffer besides, however, from symptoms that are more purely psychoneurotic. They have dreads and doubts of various kinds, usually one dread or doubt being more prominent than the others, but with their condition seldom limited to a single doubt or dread. They are all prone to get extremely tired toward the end of the day, but are not infrequently very tired in feeling at least when they wake up in the morning, especially in damp or changeable weather. They complain of a very discouraging sense of inadequacy for their work. Sometimes they become extremely diffident so that their work is less efficient than it should be, and many of them have the feeling that a breakdown or nervous exhaustion, as they like to call it, though of course the very term itself has an unfavorably suggestive element in it and should be mentioned to patients only with caution, is impending. Going about their work with the thought that a nervous breakdown is probably not far off, they have two sources of expenditure of energy—their work itself and worry over the consequences whenever they do anything. Many of them come with the ready-made diagnosis neurasthenia, and some of them are undoubtedly sufferers from what would probably be called psychasthenia; though all of them that I refer to have one symptom in common, they are very much under weight. Commonly this lack of weight amounts to more than ten per cent., but in not a few of the patients it is actually more than twenty per cent., so that it is no wonder that they feel a lack of energy. They actually do not possess the physical power to go on with their work. To use a homely figure not enough fuel goes into their boiler to feed the engine for its work.

These patients rather readily lose in weight whenever they are much occupied and above all if there are any special worries associated with their occupation. On the other hand, most of them gain in weight rather readily—and it is not an unusual thing to have them gain twelve to fifteen pounds in a vacation of two or three weeks—and then they feel ever so much better. Those of them who are school teachers begin to run down in weight almost as soon as they get back to school, recover a good deal of it during the Christmas vacation, but are rather played out at Easter time and do not gain so well and feel almost at the end of their tether by the close of

the year. After a time most of these patients find that their vacation does not do them so much good as before, and then they begin to feel, very often, that there must be something of a serious nature constitutionally the matter with them. Occasionally they have the feeling that their work is too hard for them or the strain on them is too great and that they will have to give up, and this becomes a new and poignant source of worry for them, especially if they have people dependent on them. Altogether they represent some very interesting, though rather pitiable cases; and it has always seemed surprising to me that the question of their weight was not taken more seriously into consideration by their physicians.

Many of these patients have been treated by all sorts of new-fangled remedies and occasionally have been benefited temporarily by them, the benefit received being always, so far as the history can be secured, exactly commensurate with the gain in weight. Twenty years ago they had usually been treated, before I saw them, with liberal doses of the hypophosphites and sometimes with cod-liver oil with temporary success. Now that we have found that there are vitamins in cod-liver oil it is easier to understand its tonic as well as its nutritional quality, but then plain ordinary cream has an abundance of vitamins, is almost as nutritious as cod-liver oil and is, almost needless to say, ever so much easier to take. Ten years ago a few of these patients had been treated by the cacodylates and by certain of the coca preparations following the French school of therapeutics. In recent years glandular therapy in some form has been used on them, sometimes with passing success. During this last year or two various injections sometimes of gold and silver salts and of other rather expensive drugs have come into the history. Injections of one kind or another, however, are manifestly the favorites in therapy for all the vague neurotic cases which come for treatment in our day.

We are likely to hear so much just at the present time about the value for such cases as these of glandular therapy endocrine therapeutics, to use the satisfying long Greek terms in which, to quote George Eliot, we map out our ignorance, that it has been interesting to follow up some of the cases in which apparently a number of indications for glandular therapy could be found and note the successful results to be obtained by so simple a means as food. My experi-

ence is now large enough with this class of patients so that I cannot help but think that a great many patients who are reported as cured by some form of ingestion of glandular products would have been just as much benefited if only their glandular system had been made to do its work better than before by receiving a proper amount of nutrition. I have no doubt at all that when a patient is twenty per cent. or more under weight his glandular system fails to work as it should and undoubtedly a vicious circle is formed in which the glands not receiving the nutrition which they require fail to secrete the substances which are most precious and indeed practically invaluable, if not absolutely necessary, for the proper conduction of processes of metabolism. Gland therapy, if given at the same time with encouragement to eat, would undoubtedly be followed by improvement in health, but the question to my mind would be whether this improvement was not due more directly to the increased nutrition than to any specific glandular therapeutic effect. Certainly the food therapy ought to be given a thorough trial in these cases as a sort of therapeutic test before any more specific treatment is applied.

Almost needless to say, it is extremely important for the physician to assure himself as a preliminary to any treatment of patients of this kind, that there is no definite organic stomach lesion, such as cancer in the old or ulcer in the young, to complicate the case. As a rule these are not hard to eliminate in the class of patients that I refer to. There must be neither abnormally high nor abnormally low blood-pressure, for in either of these conditions a sense of mental incapacity and headache with loss of appetite may be among the very early symptoms which send patients to a physician. There must of course be no albumin, or if a trace of albumin is present there must be no granular casts. I have seen a trace of albumin with hyaline casts disappear under proper, that is reasonably abundant, feeding. The condition was probably due to blood poverty. Sugar must be carefully looked for because in a good many cases the first symptom for which a diabetic patient applies to a physician for treatment is tiredness or achy muscles with a sense of inadequacy for his work. If there is any cough special attention must be paid to the lungs for it must never be forgotten that many a case of so-called neurasthenia is really incipient tuberculosis.

Where marked psychoneurotic symptoms are present the family history must be carefully gone into for insanity. Many a so-called neurasthenic commits suicide in a state that is true melancholia. In most of the cases that I am discussing in this paper the fact that the patients had made the rounds to a series of physicians, often carrying the records of examinations of urine and sputum with them and sometimes also the report as to their blood-pressure, simplified the problem of diagnosis, though even with all these, at times personal examinations showed quite unexpected, but very definite pathological conditions. It must never be forgotten that tumors of both brain and spinal cord often present what seem to be neurotic or distinctly hysterical symptoms at the beginning.

The cases that I refer to as bettered by gain in weight are such as nearly every physician is familiar with. They are the functional neurotic cases and their name is legion for there are a great many of them in this country. They are the stock in trade of all sorts of irregular practitioners. They constitute the material on which is based the evidence for the cures that fail, which like the poor, we have always with us. Anything that will tempt these patients to eat will improve them. Probably nothing would do more to lessen quackery in this country than if the regular physician could be brought to see the necessity for just simple increase in nutrition for these cases.

*Food Dreads.*—In many of these patients suffering from nervous symptoms, panicky about themselves, not sleeping well at night, dreaming a good deal, waking in the early morning and then not getting to sleep again, complaining of tendencies to headache with occasional half-sided headache, though not typical migraine, there is actually a series of food dreads. They are the kind who accumulate the "don't's" rather than the "do's" of life. From their reading and the suggestions of friends and the advice of physicians as well as from what they think is their own experience, they come to eliminate a great many of the most valuable articles of food in their diet. It is surprising how many of these for instance have taken all the citrous fruits out of their diet because there was acid in them and for some reason they were sure that acid did not agree with them, and they have eliminated tomatoes for the same reason and not infrequently turnips and carrots because they considered them to be coarse vegetables and rather indigestible. Parsnips they would not think of, sweet potatoes

and bananas are taboo because their consistency makes them difficult for the gastric juice to penetrate, and so their permitted food list becomes very narrow.

Not a few of them foster ideas also with regard to microbic disturbances, and they are very much afraid of germs. As a result they take very little raw food. They may occasionally indulge in a little raw lettuce and celery, but that indulgence is not without a good many qualms of their sanitary conscience. As for the idea of ever eating raw cabbage or raw turnip, that of course is out of the question entirely for them, and raw apples are likely to be looked upon as in the same category or entirely too crude for their digestion.

It is easy to understand, then, that they are eliminating most of the vitamins from their diet and as now in practically all of the larger cities our milk is supplied to us pasteurized, there is comparatively little chance for them to get them in sufficient quantity for systematic efficiency. Vitamins probably represent the best tonic for metabolism that we have, but to obtain them there must be a number of raw articles in the diet. The one article above all that may be served cooked and still supply vitamins—tomatoes, is avoided by so many of these nervous people particularly, as to make its employment by those who need it most almost out of the question.

Very often these patients, however, not only dread certain varieties of food but they dread the eating of such a quantity of food as is absolutely necessary for their proper nutrition. When they take cereal in the morning it consists of a single tablespoonful, or at most two, of oatmeal or perhaps a small sauce dish of one of the dry, ready-prepared cereals, whose food value is so small in the quantity taken as to be almost negligible though of course the cream taken with it may be very valuable. When these patients are told to take a liberal helping of oatmeal at least half a dozen tablespoonfuls, preferably in a small soup plate and then to take a lump of butter, place it in the middle of the plate and heap the oatmeal over it until it melts and then cover it with a layer of sugar and cream, they feel sure that it will play havoc with their digestion. I have seen literally hundreds of cases in which patients found to their surprise that, instead of feeling more uncomfortable after what at first they felt like calling "mess like that," they were much more comfortable after their breakfast, and above all after a few days they

found that they did not have to take laxatives in the way that they had been accustomed to do for years before.

It is extremely important as a rule to be quite specific in the directions as to eating for these people and literally to prescribe what you want them to eat and how much. Oatmeal taken as suggested, after half a grapefruit for the sake of the vitamins but eaten with all the pulp so that at the conclusion the grapefruit rind is quite cleaned out—this for the sake of the residue thus supplied for the stimulation of intestinal peristalsis—followed by a couple of eggs and two or three slices of bacon with perhaps some marmalade and toast makes a breakfast that most working people ought to take. This is quite as true for the indoor worker as for the outdoor worker, though of course the bacon must be increased in quantity for the outdoor worker. To a great many nervous people who are secretaries, stenographers, bookkeepers, and the like, *that* seems almost an impossible breakfast, but it only requires a little insistence and training to get them to take it and it will change the whole complexion of the day for them once they get used to it. They have a sense of adequacy with their work and have energy to spare that they did not have before; their irritability is lessened, and their power to stand all sorts of troubles without wincing under them increases.

Of course they will put on weight under this regime very soon, but many of them need it for there is nothing like a proper amount of weight to dampen nerves, and to borrow an expression from acoustics, to keep them from vibrating too readily and responding too violently to emotions or worries. Our English word *buxom* has a very interesting etymology that illustrates that. In old high German it was *biegsam*, that is bendable or readily yielding, hence easy to get along with, obedient. The word in English gradually changed to the form *buxom* and then came to have the meaning of rounded or ample in proportion, and when we talk about a *buxom* woman we mean one that is good to look upon for her size. The connection between the two words undoubtedly is that the rounded woman is much more likely to be easy to get along with than is the thin and scrawny one. What truth there may be in this old-fashioned idea only carefully controlled observation would show, but certainly this was the impression in the older time. Caesar said that Cassius had a lean and hungry look and that of such stuff traitors were made, as if they were the

discontented ones ever ready to react to disturbances of any and every kind.

It is certainly surprising to see what a difference it makes to put extra weight on these people. Life becomes very different from what it was before; they lose not only certain specific dreads that have been disturbing them but also feelings of impending disaster or as if something were sure to happen to them, a sense of something hanging over them which has often made life far from pleasant for them.

The one physical improvement which putting weight on thin people practically always brings with it is to cure the constipation from which they have often suffered for years. Personally I always have the feeling that no thin person should ever be given laxative medicines until after a very definite effort is made to increase the amount eaten so as to be sure that there is sufficient residue left properly to stimulate peristalsis to carry on its activity. No wonder that the bowels get sluggish when they have but very little work to do. Over and over again I have seen them respond finely and naturally just to a reasonable amount of food without any need for irritant or stimulant drugs. I make it a rule to try this out very thoroughly and explain very simply to patients that the best way to make material move out regularly at the other end of the gastro-intestinal tract is to see that sufficient food is put into this end of it. Of course the food should be of a kind that carries a good deal of residue with it. All that is so much insisted on that it need not be mentioned here.

A good many of these patients have a rather definite dread connected with the taking of more than a very small amount of food whenever they suffer from a tendency to constipation. They have the feeling that residual material is accumulating within them and that this contains excrementitious substances that are really poisons and that they are absorbing these. Not a few of them have learned the phrase, intestinal auto-intoxication, and they roll it under their tongues as a very delightful morsel of information with regard to themselves. They limit their foods, eliminating acids and uncooked materials in their diet, and so there is no wonder that they require regular doses of laxative medicine to keep their bowels working at all. This dread has got to be faced directly, overcome by the contrary habit of eating freely and after a time their bowels will regulate themselves, and their appetite will get back to normal and not be suggested away from



them by all that they have heard and think that they know with regard to themselves. Of course when constipated persons are already stout, and care has to be taken not to add to their weight, but to bring it down some, then laxatives are needed, but fortunately nature has supplied in the various salts, remedies that serve very well for this, and these act so mechanically as to be useful for long periods, if necessary.

*Mental Incapacity.*—It is often surprising how much can be done for a man who has been under weight for years and has suffered from a whole series of symptoms that have made life anything but pleasant for him while he was under weight by simply bringing him up to weight. I have in my records the story of a man who would work for two or three months at most, usually losing some in weight all the time that he was at work, and gradually getting to a point where it seemed impossible for him to go on and then he had to give up his job. When I saw him first he was about five feet five in height, weighed a hundred and fifteen pounds, was eating very little and not sleeping very well, and having to take things constantly for his bowels. He was thirty-five years of age, but he looked older than that, and there was even a suspicion of some thickening of his arteries—more than normal. On the whole it seemed a rather unfavorable case on which to try the simple remedy of making him eat more and having him put on weight, but at least it was worth while trying so long as he was under observation and care would be taken to see that no pathological condition developed under the larger diet which it was suggested that he should take.

He was a typical instance of food-dreads for he had been to see a number of physicians, each of them had counselled him to remove something from his diet, usually two or three varieties of things. Once inhibition began it was not lifted formally so now he was taking cereals, cream, not much meat, no fat, no raw fruit and no bread except as toast, and no beans, though peas were allowed. He was living very largely on such carbohydrates as rice, potatoes and the simpler puddings, with eggs and milk. When it was suggested that he should forget entirely for the time being all the directions that had been given him with regard to various articles of food and just eat anything that he cared for and above all what he cared for as a boy, he was quite sure that it would not be long before his stomach would rebel against any such dietary indulgence as that. It gave him

some confidence to call his attention to the expression of a distinguished American gastrologist who declared that stomach patients either had cancer or ulcer or they had nothing in their stomachs; that the old idea of dyspepsia or indigestion as an entity in itself had practically been abandoned, and that while there were a good many stomach symptoms apart from cancer or ulcer these were due to worries and mental conditions, but above all to the fact that a good many people did not give their stomachs enough to do.

He had an excellent sense of humor, though a number of these digestive patients have not, and so I reminded him that a well-known French physician declared a few years ago that much more of the indigestion that he saw was above the neck than below it. In a word it was largely dependent on states of mind. An American colleague had capped the Frenchman's expression by the formula that "there is only one thing in the world that is really indigestible and that is the human mind. Whenever you get that on your stomach you have got to lift it off for you cannot digest it and it seriously interferes with the digestion of other things." After some little persuasion—for he had been in the hands of many physicians—I succeeded gradually in having the patient take a good liberal breakfast, luncheon and dinner and then a cup of milk and some crackers or a piece of simple cake on going to bed. He was sure that this would disturb his sleep but very soon found that it had exactly the opposite effect, and that he never slept better, and especially slept through until he was waked in the morning by his alarm clock when he took a snack shortly before going to bed.

He began to gain in weight at once and with almost no stomach disturbance of any kind. Once at the end of about two weeks he had a feeling of nausea after eating; this recurred after dinner the following day, but he was rather inclined to think afterwards that it was due to his having eaten onions which always caused him some little stomach disturbance, even from boyhood, than to anything more serious. He gained five pounds in two weeks, was able to give up taking laxatives which he had been accustomed to take for years, and felt a marked gain in strength and capacity. After a certain amount of continual effort of mind he had always felt for years as if there was danger of his over-doing mental work and causing some serious breakdown in mental operations. He gradually lost that completely,

and after he had gained fifteen pounds in five weeks was advised to go back to work for the sake of the occupation of mind that it would afford. For the first time in over ten years he went to work a whole year through without a break. By insisting on something more than a mile of a walk on his way to work in the morning and at least as much on the way home and prescribing several hours of outing on Saturday afternoon and Sunday, he was able to go on with his gain in weight until he weighed 140 pounds.

Almost needless to say this has made him look much younger and the compliments of his friends and acquaintances on his appearance made a series of most favorable suggestions for him. He began to feel ever so much better than before and above all to lose a certain feeling of mental incapacity which had kept him all of his life so far in a lower clerkship, though he had no little ability for the management of business details as was proved during the war when he was advanced rather rapidly in official position and salary because of the number of men who had been taken from business by the war. He has continued in good health, and though his blood-pressure is probably a little higher than it should be, running between 150 and 165, and his arteries are probably a little thickened, he is getting ever so much more out of life than before. His advance in position, while it has added something of responsibility, has made his work really easier so far as physical effort is concerned and has enabled him to have more time to himself and to secure such recreation and outing as probably mean more for relaxation and lessening of the tension of nervous irritability than before.

*Backwardness, Shyness.*—There are a good many people who in the modern rush of business feel themselves unable to keep up the strenuous active life which seems to them at least to come so natural to others and who have to make a great effort to keep up with their occupation, especially when that involves frequent contact with strangers. To be asked questions makes them so nervous that they are unable to respond readily, even when they have the information all at hand. They rather easily get mixed up or confused or at least get a certain aphasia which makes them feel very foolish. It is surprising how many of these patients can be benefited by so simple a procedure as making them gain in weight. Whenever one of these patients is already up to weight I know that the question of benefit is going to

be very difficult to answer and usually will require a good deal of training in self-discipline and in deliberately forming contrary habits. When they are under-weight, however, as they are very frequently, I know that almost as a rule without exception, all that is needed to benefit them a great deal is to see that they get up to weight, and to put into their minds the suggestion that as they gain in weight they will lose their shyness to a considerable extent, and will find it ever so much easier to go on with their occupation.

Some cases that illustrate this are in my records. For instance there was the young man of twenty-seven who came from a country town to work in a public office in New York which required him to answer a good many questions. It was a severe effort for him to go on with his occupation; he was so wearied from it at the end of the day that he felt quite played out. Because of the dread of the ordeal through which he would have to go during the day, he had very little appetite for breakfast, and his lunch was a hurried and scanty meal for practically the same reason—because it came in between two periods of really acute personal discomfort. The only meal that he was eating with any satisfaction was the evening meal, but he was constantly losing in weight and when ever he awakened in the morning, no matter how early it was, he was sure to stay awake because of his dread of the day to come and the disturbance of mind that it entailed. He was losing in weight and under the circumstances it is not surprising that his symptoms were getting worse and worse; his appetite was going; he was losing more sleep, and a breakdown seemed imminent. He had been given the hypophosphites, the glycerophosphates and a course of quinine, iron and strychnine, and obtained no relief. He had certain gastric symptoms from the presence of gas—for there was nothing else in his stomach, and in this region at least nature abhors a vacuum—and he had been treated for nervous indigestion and intestinal auto-toxæmia and a number of cognate things.

He came to me some years ago, when as yet I had had comparatively little experience with these thin neurotics and so I feared that I would not be able to do him very much good. I made it clear to him, however, that he must gain in weight as a preliminary to any improvement that could be expected. I proceeded to dictate what I thought he should eat for each of his meals. Baked apple or a banana, a liberal helping of cereal, two eggs and some bacon in the morning, also two

slices of toast with a little marmalade, this last because he complained somewhat of constipation, and no wonder with the small amount he was eating, and I have often found that marmalade at breakfast will save a pill or a dose of cascara at night. At lunch he was to have some stew as a rule and some potatoes, usually some string beans, or some form of greens, and then some dessert; he was shocked when I suggested pie. Instead of coffee and tea I recommended that he should take hot milk with a dash of coffee or tea for flavoring. It is surprising how satisfying just the taste of coffee is in the morning, provided the milk is heated. Coffee is after all only an aroma and not a real taste. I once had a woman patient who as the result of severe diphtheria in her younger years had completely lost her sense of smell yet who thought very much of her coffee and felt that she could not get on without it until I demonstrated to her that she could not tell the difference between warm water with milk and sugar in it and strong coffee with similar additions when she was blindfolded. People who crave coffee and feel they could not get on without it in the morning in the course of a week find that they get on with *café au lait* quite as well as if they had the strongest coffee.

For his evening meal I suggested that my patient should have some thick soup, a good helping of some substantial meat, a couple of vegetables, usually a salad and then some rather substantial dessert. He was quite sure that if he took all this he would not be able to digest it, but with an ordinary strichnine tonic and a slight gradation of eating toward the maximum required, he very soon found that he could not only digest very well, but that he actually felt much better in his insides, as he said himself, than he did before. He gained nearly five pounds the first week and three pounds regularly after that for five or six weeks. Five feet nine in height, he weighed less than 130 pounds when he came and by the time he weighed 150 pounds realized that he was sleeping better, and that he was having ever so much less trouble in facing people who asked questions. By the time he got to weigh 160 pounds he was so much better in all his symptoms as to realize that the main thing that he needed was more physical energy in order to enable him to use his will power properly. At this stage it was comparatively easy for him to face even the prospect of being transferred to the information department

of his concern where his business would be to answer all sorts of questions all day long.

He has never quite gotten over a certain sense of shyness with regard to the beginning of his day's work. This was evidently dispositional. It had been very greatly emphasized, however, by habits of yielding to his feelings, and then unfortunately exaggerated by his lack of nutrition which left him without such will power as would have enabled him to overcome the tendency that was natural to him. He is quite convinced personally that the way to cure all the dreads is to put weight on people but as I have a number of patients in my records who have dreads who are not only up to their normal weight but even well beyond it, I know better than that, but the experience was very valuable to me in making me realize that weight alone without any other specific treatment was of very great importance for these psychoneurotic symptoms when they occurred in thin people.

*Diffidence.*—A man, aged thirty-eight, the manager of a rather important business concern with which he had been connected for some fifteen years, came to me quite hopelessly because he felt sure that in spite of the recommendation of a friend who had been greatly helped in a matter of backwardness, that *his* condition was due to natural disposition and probably could not be helped. He had been offered a district managership for his company at a distance from home with a salary once and a half what he was getting and an opportunity for advancement to double his salary if he made good in the new position in the course of three years, and had refused because he found it so hard to meet new people and was so disturbed by the thought of having to face introductions to strangers or much occupation with any but those with whom he was quite familiar. He had practically given up going away on vacations because of the discomfort occasioned by having to meet strangers. The worst of it was that as his family did not like to leave him alone this had kept them in town for several summers, and now his ailment was taking the form of making him refuse invitations to social affairs of almost any and every kind, even though he might be reasonably assured of meeting only those whom he knew quite well. His symptoms had been progressing for years, and he was yielding to his dreads rather than opposing them. There was some question whether there was not a melancholic

element in the case, though there was absolutely no family history of any mental trouble.

The one hopeful feature was that though of good muscular build, indeed an athlete to some extent when he was younger, he was very much reduced in weight. One reason for this indeed was that he had only a limited time for lunch because the middle of the days was the busiest time in his day's business; he hurried through a glass of milk and a sandwich or sometimes a piece of pie because he did not like to be in the room where so many strangers were with him at the lunch hour. Once or twice a week he went to a little dining club where he met old friends and then had a good lunch, but that always took more than an hour and he did not feel that he could afford the time. Five feet ten in height, he weighed just under 140 pounds, and had been coming down in weight, slowly but surely for several years.

He was told very candidly that the one thing that I could surely do for him was to put weight on him if he would only follow directions, and when he had gained thirty pounds in weight we would then see what was left of his symptoms and endeavor to treat those specifically. He had been eating a very small breakfast, coffee and a roll, but a very substantial dinner at night, probably more than was actually good for him. He was falling asleep in his chair afterward, felt miserable of course when he did so and was usually spending some nine hours in bed. He slept quite well but felt that he did not get enough of sleep. It is surprising how many people who do not get out in the air much, sleep for eight or nine hours and yet do not feel rested and are under the impression that they ought to have more sleep. Unless there is some very good reason of a special nature no ordinary human being above the age of twenty ought ever to be allowed to be in bed regularly more than eight hours. He accumulates energy that as a rule he cannot use up the next day and as we have no accumulator in the human body his surplus energy is used up in work within the machine itself, usually with the production of all sorts of unpleasant feelings.

This patient needed to have more exercise put into his daily life, and I insisted on his walking to his office, two miles and a half, and walking back in the evening, except in the very worst weather. It took him only twenty minutes longer than to take the subway and instead of the closed, confined car during the rush hour, jostled by

the mob standing on his feet, swayed this way and that, and especially in the evening feeling extremely disturbed as a consequence, he got home for a bath and a rub down just before dinner feeling thoroughly fit and ready for his meal.

After dinner he was not permitted to sit down for more than an hour, but then had to go out somewhere for a walk or to visit a friend or to a lecture—for the movies did not appeal to him and always gave him an intense sense of being crowded in with others. Before going to bed he had to have a glass of milk and some crackers or a piece of cake, usually a rub down with water followed by alcohol just before turning in for the night. It was rather hard to get him to eat all that was necessary. He gained the first week, then remained stationary, gained again when his attention was particularly called to it, but had to be constantly bucked up to keep him at his eating. It took six months to gain fifteen pounds, then he was in a railroad accident in which a number of people were killed and promptly lost ten pounds. The task had to be begun all over again. In a year, however, he had gained twenty-five pounds. He noticed how much better he was able to get along with people and then was encouraged to eat heartily and now after five years he has the habit of eating; he weighs 170 pounds and I prefer not to let him gain any more. His own feeling is that he has been immensely benefited. There has been absolutely no mystery about it, however, for I told him very simply at the beginning that I wanted to put weight on him, so now he feels no dependence on drug or doctor and knows that he must just depend on himself.

The more I see of these patients the more I am persuaded that what a great many people need is not so much drugs or remedial measures as health engineering. They need to have their lives taken apart from early morning until the next morning and all the frictions from misunderstanding, dreads, unfortunate advice and suggestions from all quarters removed and their habits made over again. It is surprising how many of them can be greatly benefited by this means. I have had them at all ages—from seventeen and eighteen up to sixty-five—improve very much simply as the result of seeing that they got enough to eat. There are many more people starving themselves either in quantity or quality than is usually thought. A great many of the thin neurotics are just suffering from nerve



starvation. They are getting cured by all sorts of means, from Eddyism to Freudianism, but any suggestion will cure them that makes them eat better.

It would be immensely valuable to these people to get them to realize that their lowered state of nutrition meant more than any other factor for their neurotic and psychoneurotic symptoms. It is true that these are usually dispositional and are founded deeply in the nature of the individual representing distinct idiosyncracies, but they can be overcome by contrary habits whenever the individual has sufficient physical strength and surplus energy to devote to that purpose. They cannot be made to disappear entirely, and constant and persistent efforts will have to be made to keep the tendencies to them in check, but the neutralization of these tendencies is ever so much easier when the individual is up to weight. Once this has been demonstrated to him, he feels entirely different toward his symptoms, for this experience puts them on a very definite material plane and does not leave them the vague forces for disturbance which often are so unfavorably suggestive and keep the patient from having that confidence in himself that will enable him to do things.

For many physicians, doubtless, the treatment will seem to be entirely too simple to be efficient. As a matter of fact the real therapeutic factor in a great many different kinds of ailments that have been cured in the past by all sorts of remedies has been exactly the increase of nutrition. The most important chapter in the history of medicine is the chapter of the cures that have failed. All sorts of remedies and modes of treatment have cured for a time and then proved ineffective, but the confidence in them has given a better appetite and that has done the work in many cases.

In order to get people to eat and keep them from having the fatigued feelings of their gastro-intestinal muscularis because the muscles have to deal with so much more feed than before, strichnine is the all important indication. I prefer to give it in the form of nux vomica because I think that the tincture contains certain principles that are not in the alkaloid that are very valuable. I give a little more than one drop for every ten pounds of weight. Beginning with fifteen drops for those 130 or over and gradually increasing to twenty or twenty-five drops three times a day before meals. Combined with tincture of gentian, and tincture of cinchona, as sto-

machics, for the sake of the appetizing effect, it is usually not hard to get people to eat. The word of command in this matter and the will to eat is more important, however, than any artificial appetite that can be created. After a little persistence in eating, a habit is established, and then people find it hard to abstain from food as they did before, for they have a craving for it three or four times a day. Once gain in weight of ten per cent. is secured, the nervous symptoms have usually subsided to such an extent as to make the rest of the treatment plain sailing.

# Pædiatrics

EDITED BY JOHN FOOTE, M.D.  
Washington, D.C.

## DISTURBANCES OF HEARING IN CHILDREN AND THEIR INFLUENCE ON THEIR DEVELOPMENT

By PROFESSOR DOCTOR VICTOR HAMMERSCHLAG

English by B. Lewis.\*

It would be well to call to attention a matter hitherto but little regarded, the influence exerted on the further development, both physical and mental, of children affected by disturbances of hearing. This is not always evident as such disturbances often do not proceed from a painful affection of the ear and therefore remain unnoticed. Such an unnoticed disturbance of hearing is the more serious as it interferes with the child's mental and physical development from a cause which is not known and therefore cannot be counter-acted. Although such a disturbance may exist even in infancy it is usually not discovered before the child goes to school. When there, bright and intelligent children, thus afflicted, are thought to be dull or inattentive because they cannot answer to the teacher's questions which, of course, they do not fully hear.

Such disturbances of hearing I would divide into two classes, curable and incurable. The curable ones should be treated medically, the incurable ones pedagogically.

I shall begin by explaining what is meant by hardness of hearing. It is not quite easy to do so scientifically, although ordinarily the term is well understood. For the purpose in view I would state the meaning of the conception of hearing as a qualitative and a quantitative one. This division corresponds to the two physical attributes of the simple sound, the *quality*, that is the height of the sound (number of vibration), and the *quantity*, that is the intensity of the sound (extent of vibration).

As regards the qualitative perception of sounds, it is hearing the different sounds without regard to their loudness. This includes twelve

---

\* Wien, XIX Vegagasse 15.

octaves, from the lowest that can be perceived with eleven double vibrations, to the highest sounds with about fifty-five thousand vibrations a second.

According to the doctrine of Helmholtz and Hensen, the cochlea in the human being is a kind of harp with transverse vibrating strings of various length and pitch corresponding to the various sounds; the basal membrane of the cochlea consists of these strings. When irritated they carry the sound to the sensory cells of the organ of Corti, situated on the basal membrane, and thence to the brain in the path of the cochlear branch of the auditory nerve. From a physiological process like this it is evident that entire deafness must follow if by some disease of the cochlea the organ of hearing becomes destroyed, and now no single fibre of the basal membrane capable of vibration, no single irritable sensory cell, no single terminal nerve fibre, remains intact. But if a part of the perceptive organ remains preserved, those sounds are heard which correspond to that particular part, in brief, *the person in question is no longer wholly deaf, but has a hearing island at that spot.*

We may now proceed to picture to ourselves how a partial disturbance of hearing originates: The integrity of the sensory epithelium is injured at any place of the basal membrane, but its activity does not quite cease. Then, though the *qualitative* capacity of hearing is present for the corresponding sounds, the *quantitative* capacity of hearing is lessened.

The stimulus-threshold for these sounds is higher up; the sounds must be louder and stronger to be heard; the patient is hard of hearing for the sounds corresponding to the injured part of the basal membrane, or to the nervous terminal apparatus, or to both organs.

We have mentioned those disturbances of the qualitative and quantitative capacity of hearing which come about in consequence of affections of the sound-perceiving apparatus. Similar consequences are found in the various affections of the sound-conducting apparatus; namely, of the middle ear and its air-containing accessory spaces. As a general thing, all affections of the middle ear cause disturbances of hearing for the reason that a pathological rigidity of the auditory ossicle chain follows. The clinical picture shows this rigidity principally in a diminution of hearing for the deep and

deepest sounds; this because they require on account of their great number of vibrations more particularly a normally vibrating sound-conducting apparatus. Therefore, if an obstacle to sound-conduction be present the deep sounds are heard with great difficulty or not at all.

We see that what we generally call hardness of hearing consists clinically of three forms.

1 The perception within the range of a part of the human sounds is entirely extinct.

2 The perception within the range of a part of the human sounds becomes more difficult.

3 The perception within the range of a part of the entire human sounds becomes more difficult.

The fourth possibility that the perception within the range of the entire human sounds is dead need not be considered here, for this does not mean hardness of hearing but entire deafness in the child.

Later it will be evident that the matter is not so simple, for in general we do not hear merely simple physical sounds, but in most cases compound sounds and various noises.

Clinical material shall now be the test for what has been said about the deficiency of perception for various sounds.

For this purpose we shall take as material those children so hard of hearing as to be called deaf, and accordingly be placed in asylums for the deaf and dumb, yet still retaining some residues of hearing.

As is well known, some deaf-mutes still have a "slight capacity of qualitative hearing left. Indeed, in the majority of cases we have to deal with some greater or less deficiency in the qualitative capacity of hearing and, moreover, with an injury of the quantitative capacity of hearing for the sounds qualitatively preserved.

Urbantschitsch was the first to use the accordion for testing the deficiencies in qualitative capacity of hearing in the deaf and dumb; Bezold afterward employed a series of tuning-forks for the purpose.

In regard to the Bezold test I would merely say that with this apparatus of tuning-forks every perceptible sound can be made to come to the hearing free from all accompanying sounds.

Tuning-forks are used for the deep sounds, and small covered organ-pipes for the high ones. Bezold tested the hearing of the children in the Munich deaf-and-dumb asylum with marked success. Other specialists having undertaken similar tests, we now have a large amount of material for the purpose of judging the capacity of hearing on "deaf-mute" children.

We will now proceed to sum up these results. To start with, I must state that there are many deaf-mutes who are absolutely unable to perceive the faintest sound; these children are actually deaf and dumb. Others retain remnants of tone perception in the form of small and tiny tone islands. There are children, for example, who are able to hear two or more adjacent tones from some range of sound, and we can naturally not call such children deaf, and even less those children who possess several such tone islands with large spaces between.

We find a greater capacity of sound perception in such children as show two larger tone paths with a small gap between. There are children also in whom the range of tone perception is connected with but a shortening at either the upper or lower end or at both ends. This shortening may become so much smaller in some children that, qualitatively, their range of hearing hardly differs from that of the normal range.

We must now ask in what way these children differ from children with normal hearing. The answer is only in the quantitative capacity. A child with normal hearing hears every perceptible sound at its slightest stroke, whereas a child hard of hearing hears a sound for which it has perception only upon the strongest stroke, and then only for a very short time.

For determining the quantitative capacity of hearing of such children we must note the duration of hearing of the tuning-fork sounds they can hear. Nager states that this duration is only a small fraction of the normal length of duration. Yet, not even in those children who hear best is this duration as long as half the normal one but only one third or even less.

We must now ask how disturbances of hearing become manifest in ordinary intercourse. Let us regard Itard's old classification of this affection. At the deaf-and-dumb school in Paris he classified his material into four groups, according to possible perception for

sounds, vowels, words and sentences. In my experience and that of other otologists we rarely find children with affections of the inner ear whose hearing reaches farther than one half to one metre for a few connected words uttered in a loud voice. Of course, such children cannot learn to speak by ear. They are therefore mostly found in schools for deaf-mutes where they learn to recognize words from the motion of the lips.

We shall now turn to those children with a medium degree of hardness of hearing not easily recognizable to persons who are not professionals. To trace the causes of such disturbances of hearing the following pathological processes of the ear must be examined.

1 *The suppurations of the middle ear* which ensue after acute exanthematic infectious diseases in childhood. It is generally known that the suppurations of the middle ear occurring after scarlet fever are the most important. Such suppurations frequently last for years, and occasion distinct disturbances of hearing.

2 *The catarrhs of the Eustachian tube and tympanic cavity* with which children are so often affected. These are caused by chronical conditions of swelling in the naso-pharyngeal mucous membrane, and especially by the hypertrophy of the lymphatic pharyngeal ring (hypertrophy of the pharyngeal tonsils and adenoid vegetations). You are all familiar with the picture of the so-called tubal catarrh. A lesser or greater contraction of the tympanic membrane is seen while, at the same time, it is shiny and yellowish in color because of the serous transudate in the tympanic cavity. It is very characteristic how the disturbance of hearing varies. Whenever the chronic condition of swelling of the naso-pharynx becomes more acute (in early spring or late autumn when the weather changes the hardness of hearing increases much. Then again we observe that it decreases and the child is able to hear the teachers words. No doubt it is this variation which causes parents and teachers and even physicians to overlook this infirmity. To cure this condition fully it is absolutely necessary to remove the cause, namely to cut out the pharyngeal tonsils and the adenoid vegetations.

3 *Disturbances of hearing of medium degree* in consequence of affections of the sound-perceiving apparatus, that is, the inner ear. We have such cases very rarely, for affections of the inner ear in childhood usually cause such severe disturbances of hearing that the

children so afflicted are mostly found in the schools for deaf-mutes; therefore we need not consider these cases here. However, disturbances of hearing of medium degree—which I must call incurable—may be caused by congenital abnormal developments of the inner ear. I have often observed that in families where deafness is hereditary one child may from birth only have a disturbance of hearing of medium degree.

It has already been said that the children hard of hearing because of a labyrinth affection are very rare and therefore need no mention here. Let us therefore take into consideration only those children afflicted with suppurations and catarrhs of the middle ear. Such children show very characteristic deficiencies in qualitative capacity of hearing when tested with the Bezold tuning-forks; for the reasons mentioned the lower portion of the range of sound is absent. The affected ear does not perceive the sounds of the tuning-forks of medium height as well as the healthy one; namely, the gradually expiring sound of the tuning-fork ceases sooner than in the normal ear. (The time of hearing is diminished; the quantitative capacity of hearing is impaired.) The hearing-distance for speech is correspondingly lessened, for example, a whisper which is clearly heard by children with a normal hearing at a distance of twenty to twenty-five metres or even more is heard by afflicted children either only *ad concham*, or by less afflicted children at a distance of one or two metres.

It is difficult to determine how great a disturbance of hearing must be in the infant to allow us to say that it must be mentally impaired thereby, but we can say that no disturbance of hearing in childhood is so small that it may not impair the acquirement of speech.

We must now reply to the following question: In what degree does a child's development suffer when impaired in hearing and what is it that gives rise to such disadvantages?

As a certain specialist correctly remarks, a person hard of hearing may be said as of vision to be near of hearing that is to say, he perceives the sound later in the degree as its source approaches him and when it moves away he ceases to hear it sooner than the normal person. In addition the person hard of hearing is also weak of hearing; he fails to hear various sounds however close-by whenever the irritation is not strong enough.



In conclusion the perception of hearing still present is reduced in strength and changes qualitatively which is the chief point. One who is hard of hearing hears sounds which are not only changed but distorted as well. A child who perceives only such distorted images of music and other noises loses the most important faculty in childhood—that of acquiring speech.

I shall proceed to present this more clearly. The tones of our musical instruments are not simple physical sounds, but tone combinations of the fundamental tone with the various harmonious upper tones which vary in the different instruments. It is the combination of these upper tones which gives its character to the musical tone so that, for example, the  $a^1$  of the violin sounds differently to that of the flute. It is easy to understand that in order to distinguish all these higher tones we must have a normal hearing function; naturally it must otherwise happen that we miss some of these higher tones which barely reach above the stimulus-threshold; we therefore hear an altered tone.

We have herewith mentioned only one circumstance which causes defects of hearing for musical tones. It is evident that such a failing beginning early in childhood must impair the musical perception of the child.

The same is true of speech also which consists of sounds and noises. Helmholtz and Donders were able to discover by their examinations what pitch of the tone corresponds to the single vowels.

According to Helmholtz the vowels in the scale are designated as follows:

	corresponds to the vowel U
f	
I	
b	" " " " O
II	
b	" " " " A
III	
cis	" " " " Ö
III III	
g - as	" " " " Ů
III	
b	" " " " E
IV	
d	" " " " I

Wolf found the same for the consonants in so far as they are self-sounding (R-B-K-T-S-F-G-Sch).

Wolf and Helmholtz define a noise as a succession of simple tones which are partly very near each other in the scale and are mostly subject to a very quick and irregular change of various short tones.

We may therefore designate noises—in our case the consonants—as musical as it were. The following table shows how they are inserted in the scale.

R	C-2	+ C-1	+ C+c
B and P	<sub>a</sub> I		
K	<sub>a</sub> II-	<sub>a</sub> III	
T	<sub>aa</sub> II-	<sub>aa</sub> III	
F	II-	<sub>a</sub> III	
S	<sub>c</sub> IV-	<sub>c</sub> V	
Sch	<sub>aa</sub> IV+	<sub>a</sub> IV + <sub>a</sub> III	

As we have now shown that the single lingual sounds are combined tones which can be musically determined, it is evident that single sounds will be missed by persons hard of hearing in so far as this is more or less impaired; he will therefore perceive only a confused and indistinct lingual acoustic image.

This is not all, however, the fact that a person hard of hearing is also near of hearing distorts for him this image still more and further because the various and manifold lingual sounds represent a greatly varying hearing-distance according to their acoustics.

The following table by Wolf will explain this as it shows the relation of the intensity of sound of the individual lingual sounds.

LINGUAL SOUNDS	Is STILL CLEARLY DISTINGUISHED AT THE FOLLOWING DISTANCE:
A	288 Metres
O	280 "
Ei	272 "
E	264 "
I	240 "
Eu	232 "
Au	228 "
U	224 "
Sch	160 "
S	140 "
F	55 "
M (a) and N (a)	144 " (in combination with the vowel a)
K " T	51 "
R	5 " (the R on the tip of the tongue alone)
Whispered U	40 "
B	14 "
H	10 " (by itself as intensified aspirate)

As the various lingual sounds have a various hearing-distance it is easy to understand why a child hard of hearing must miss many letters or perceive them very indistinctly. A child hard of hearing which is just beginning to learn to speak will utter the wrongly heard word equally wrongly, or not at all. In any case the acquirement of speech is made more difficult or altogether impossible. That a child's mental development must be unfavorably influenced by such a deficiency at the age when it should begin to speak is self-evident. However, later, at the fourth, fifth and sixth year an ear affection which impairs the clear perception of sounds may likewise impair the memory of speech already acquired, and such children may still suffer loss of speech. It is clear that thus at every stage of childhood the mental development may be retarded or checked. This deplorable circumstance is plainest at the age when a child should learn to speak or when it enters school.

Sad to say, the child does not only suffer in mind, but in soul as well. Kroiss gives a detailed description of this process of impairment as follows.

"An infant gradually reacts to sound impressions by winking, raising its hands, becoming startled and screaming. Its first sound impressions are extremely indistinct and confused, but gradually grow in extent. It distinguishes clearer and higher sounds out of the confused noises as it gradually comes into closer relation with the various sounds. By degrees it learns to distinguish the sources of sound, to know whence they proceed. The child is pleased at the sounds as they are connected with sensations of pleasure. The louder sounds appear more interesting and the child pays more attention to them; the faculty of listening is awakened.

"The child acquires an extraordinary number of acoustic pictures from speech. It hears only loving words from those around it. It cannot, of course, distinguish them rightly and at first recognizes among them only certain tones which express certain typical feelings. It learns the difference between words that express love from those that express displeasure. The meaning of the words it does not understand, but it knows their music."

Brauckmann points out how much is lost to the soul life of a child hard of hearing. His words are:

"Let us now consider the acoustic sensations we receive from our

fellow-beings. A child hard of hearing loses much. It cannot hear the words of love, of praise, of exhortation, of warning, of consolation, of encouragement addressed to it; the delight of a laugh, the sadness of a sob do not find the way to its soul."

I have already stated how even slight disturbances of hearing may impair the child's ear for music; we now see that it impairs its perception for speech also.

I would now add a final circumstance, the psychological fact that hearing and vision compensate one another. The disturbance of this mutual relation must needs impair the child's mental development. Let me cite Brauckmann's words:

"The ear bears certain education relations to the eye. Things we hear always seem to require optical proof. By the ear, our eyes are stimulated to action and are in need thereof. They depend on light, accomodation and direction of vision. They have to seek their irritants and must sometimes remain closed to them. It is different with hearing. Our ears constantly receive irritation from all sides, from near and far. The entire objective world speaks to them, not only that part which is bounded by our field of vision. Such guards are invaluable to the eyes. Their attention is aroused, and they receive the impetus to action. The eyes may rest in security; they are relieved by the ears, even as the eyes often relieve the sense of touch.

It is clear that the eyes of a child hard of hearing must dispense with this stimulant.

From what has been said you will see how many factors act together to the injury of a child afflicted by hardness of hearing, and we can better understand why such a child cannot answer to the acquirements of school. Bezold has illustrated in a table of figures how often this is the case. I will not repeat them here; I only wish to show you how many children are found in schools suffering from disturbances of hearing, and therefore how important it is that the physician recognizes such affection in the child.

In Riga, in 1878, Reichhardt first instituted examinations of children in school to test their hearing; the number of children amounted to 1055. For an otologist Reichhardt's examinations are not quite satisfactory as he tested only with a loud-ticking watch. Normally hearing children heard it at a distance of sixty feet.

Twenty-two and three-tenths per cent. could not hear it even at a distance of only twenty feet. Doctor Weil, of Stuttgart, tested 5905 children with a low voice, using the ear speculum at the same time. In some schools he found over thirty per cent. of children hard of hearing who were unable to hear a whisper at a distance of eight metres, while the normal distance is as much as twenty to twenty-five metres. American aurists have given us two series of examinations.

At an examination of 570 school-children by Samuel Sexton he found 13 per cent. of them to be very hard of hearing. And among 491 children Norell found 25.5 per cent. to have diminished capacity of hearing. Bezold instituted a specially close examination of 1918 children and found 74.2 per cent. to have normal hearing while 25.8 per cent. had ear affections. Six and seven per cent. of the affected children could hear a whisper on both sides at less than 8 metres, while 5 per cent. heard at less than 4 metres. It appeared therefore that 26 per cent. of the children had but one third or even less of the normal hearing-distance. The noteworthy fact about Bezold's observations is that in the higher classes of the public schools he found, on an average, less hardness of hearing, and in the high schools a very few children thus afflicted. He therefore came to the conclusion that children hard of hearing rarely continue to attend the higher grades. What is most important for us to consider is the circumstance that judging from these findings, 41.7 per cent. of the children hard of hearing could be cured more or less easily under the proper treatment. They were principally cases where the hardness of hearing was caused by accumulations of cerumen or very severe catarrhs and middle ear suppurations. Of most frequent appearance, however were the already mentioned catarrhs of the Eustachian tube which, as we know, is connected with the hypertrophy of the pharyngeal tonsil. The examinations by Stangenberg, of Stockholm, inform us how prevalent this affection is among children. He diagnosed an enlargement of the pharyngeal tonsil in 10.1 per cent. of 3244 school-children. Kayser found 920 children thus affected among 15,000 school-children. Kafemann found severe hypertrophies of the pharyngeal tonsil in 7.8 per cent. among 2238 children who had most of them been examined with the speculum.

It now remains for us to reflect in how far it is possible for the family physician to aid in this respect. We have primarily to consider the catarrhal and inflammatory affections of the organ of hearing in children. It is easy to recognize a catarrh of the Eustachian tube and to remove it. It is equally easy to remove the hypertrophy of the lymphatic pharyngeal ring which is the cause. The family physician can do even more. It is known that the hypertrophy of the pharyngeal tonsil is hereditary in some families, but the children with adenoids in most cases come too late under the physician's notice. It is only when they begin to go to school that the trouble becomes apparent. When, then, the physician is called in and recognizes the evil, he should examine the younger children as well and, if found necessary, treat them correspondingly. Greater difficulty is presented by the suppurations of the middle ear. Every child with measles, scarlet fever, diphtheria, mumps or German measles should have its ears examined by a specialist once or twice a week even if it does not complain of any disturbance. Very young children cannot explain what ails them and older children do not feel the beginning of the inflammation because of a certain numbness. An incipient inflammation of the ear can be discovered only upon systematic examinations by the physician who is then enabled to adopt the necessary treatment.

The ear of an infant particularly must be carefully watched, since inflammations of the middle ear very frequently occur in infancy. To illustrate let me say here that Kutscharianz, of Moscow, found a sound tympanic cavity in only 30 cases of 230 children who died in the foundling asylum.

What we must require as regards school-children it is scarcely possible for the family physician to comply with. It is absolutely necessary that special school-physicians should be entrusted with this matter and that they be familiar with otoscopic methods.

At the beginning of the school year he should examine the hearing and tympanic membrane of *all* the children without exception and must chiefly look for adenoid vegetations. If no school-physicians are appointed the teachers should undertake this task by testing the hearing of all the pupils for the whispering voice. Those who with *one* ear cannot hear farther than eight metres must come under an aurist's attention. For pupils, so Bruehl thinks essential,

ought to be tested several times a year, and every child upon recovery from an acute infectious disease should be at once examined by an aurist.

What is to be done after such examinations have been made? The physician must now proceed to discover whether the disturbance of hearing is the consequence of a curable affection of the ear, or whether it is incurable. Then those children who are hard of hearing would not be left to their own disadvantage in the same class with normally hearing children, but be placed in special parallel, so-called hearing classes. If the hardness of hearing is so severe as to prevent the child's being taught orally, it must be placed in a school for the deaf-and-dumb.

The question now confronts us: When shall a child that is only partially hard of hearing be sent to such an institute? Bezold holds that we should here distinguish between children hard of hearing from some cause and those who were born so. Children who are so born are not able to learn to speak from phonic images if they cannot hear ordinary speech beyond a distance of two metres and must be taught in schools for the deaf-and-dumb.

On the other hand, children who have acquired this infirmity and have already learned to speak at the time can master language the better, the later the hardness of hearing began.

From what Bezold tells us we may set up two laws:

1. Children who are hard of hearing from birth or have become so before learning to speak should be placed in a school for the deaf-and-dumb in case their hearing-distance for distinct speech does not exceed two metres.

2. Children who have acquired a disturbance of hearing after learning to speak and whose hearing-distance for distinct speech is only half a metre should also be placed in such an institution.

It is rather hard to judge when a child hard of hearing may still attend public school successfully; the child's intelligence must be a guide here. But we cannot be far wrong in demanding that such a child should have a hearing-distance of at least four metres for ordinary and not emphasized speech. Children who hear less well and cannot hear what the teacher says should be sent to school as an experiment only. As soon as it shows that the child cannot keep up with its class-mates it should be placed in the hearing class which

ought to be established in every school. Of course, it need not be particularly mentioned that the children hard of hearing should be seated nearest to the teacher. Should these children be near-sighted likewise they ought to be given fitting spectacles in order to be able to read from the teacher's lips. Fortunately children chronically hard of hearing unconsciously acquire this art of reading speech from the lips.



## PÆDIATRICS CLINIC

By DR. ROWLAND G. FREEMAN

April 28, 1921, Roosevelt Hospital, New York City

---

### SCURVY

CHILD of thirteen months, giving a history of crying when moved or diapered for the past four months, or since the age of nine months. With this, there has been noticed some soreness of the gums around the teeth that have already erupted.

The child was taken to a physician who advised the parents to give the child soups and vegetables, but notwithstanding these orders and the statement of the mother that she had administered this food, the child was admitted three days ago with spongy gums, a typical attitude of scurvy, with flexed thighs and crossed legs and tenderness on pressure of the thighs or tibia, as well as pain on movement of the lower extremities.

In addition, there was a hemorrhage into the left conjunctiva, and a marked hemorrhage of the left lower eyelid (a black and blue eye).

The blood count on admission was: Hæmoglobin, 50 per cent.; red corpuscles, 4 million; white blood-cells, 11,000; 64 per cent. polynuclear.

On admission, the child was put on orange juice and vegetables, and already the redness of the gums has disappeared; the black eye is not noticeable, and the legs can be moved without pain.

Thus, this is a typical case of scurvy which was probably properly fed only at intervals, allowing the lesion to progress. Proper feeding for only three days has practically cured the child.

These cases are rarely seen at present although three cases presented themselves at the Medical School Clinic on Tuesday last, April 26, 1921.

The first case recognized in this country was discovered by Northrup in 1889 but as soon as attention was called to this condition, cases were rapidly reported from different parts of the country so that five years later, in a collection of cases made by the American Pædiatric Society, nearly four hundred cases were reported.

## ENLARGED THYMUS

A colored boy, three years old, was admitted to the hospital with a history that for one month, he had held his breath frequently, and had had numerous convulsions during an attack of measles four months ago, but none since then.

Physical examination of this child showed a well nourished, healthy looking baby, normal head and chest measurements, scattered râles through the chest but no further evidences of lesion of the lungs.

The history of convulsions, associated with breath-holding, led to an X-ray examination of the thymus which was shown to be enlarged. The child was given X-ray treatment of the thymic region, and while the more recent pictures of the thymus gland still show some enlargement, there have been no further indications of thymic symptoms.

This treatment has been given to a considerable number of children in this service who have had either attacks of holding the breath, with stridor, or convulsions, with usually prompt relief of symptoms.

We always followed up a normal thymic shadow by X-ray. Two of these children however, after being discharged apparently in good condition, have been found dead in bed, which would indicate that the treatment in these cases had not been carried to a sufficient extent. It is difficult to determine how many treatments should be given in each particular case. If the thymic symptoms disappear and the thymic shadow becomes normal, or disappears, we may be sure the treatment has been carried far enough, but apparently a large thymic shadow may still persist in a thymus that does not contain much active thymic tissue.

## PNEUMONIA

This child, aged three years, was admitted with a history of a six day illness, with convulsions, fever, bloody sputum and cough. The fever was followed by drenching sweats. There was cutting pain in the right axillary region. Blood count showed 65 per cent. hæmoglobin; 4,600,000 red corpuscles; 25,000 white, and 83 per cent. polynuclear. Shick test was negative, and nose and throat cultures were negative. His previous history was that of an attack of bronchitis six months before.

This child was poorly nourished, looked acutely ill, had an expiratory grunt and was perspiring profusely. Physical examination showed a negative pharynx, ear drums looked dull and landmarks were indistinct. The neck was not stiff.

On examination of the chest in the right axillary region, resonance was impaired, with bronchial breathing and voice and many crepitant râles. There were coarse râles on the left side and dullness on percussion. In the posterior axillary region, there were bronchial breathing and voice. Heart was negative; abdomen distended. X-ray showed a pneumonia of the right lung, involving all but the apex. One grain of calomel was administered, and the child was put on the roof where he was kept throughout the day and night. After two days, his temperature rapidly declined to sub-normal and since then, has remained normal.

Calomel was followed by infusion of digitalis,  $\frac{1}{2}$  grain every four hours, and mustard paste.

It is remarkable how many of these cases reach a crisis within two days after they are put under this treatment. Whether this is due to the treatment or because the pneumonia has run its course, it is impossible to say.

The children however, when put on the roof immediately get more color in their cheeks, take their food better and are much less restless.

The claim that such treatment increases blood-pressure has not been well substantiated.

#### APPARENT PNEUMONIA WITHOUT FEVER OR LEUCOCYTOSIS

This child, aged eleven months, was admitted to the hospital 12 days ago with a history of paroxysmal cough, followed once by vomiting. Restlessness during the night and fretting during the day. The child had previously had a succession of boils.

On examination of the lung, there was found posteriorly on both sides, fine râles, while at the right apex, there were bronchial breathing and bronchial voice. The abdomen was retracted. Physical examination was otherwise negative.

An X-ray of the chest showed a consolidation of the right, upper and middle lobes, while in a portion of the lower lobe, areas of bronchial infiltration could be seen extending from the root of the lung.

The heart was not pulled over to the right side, appearing more as if it had been pushed to the left.

This child's temperature varied between 97.5 and 99.5. Blood count showed 65 per cent. hæmoglobin; 15,500 white cells and 39 per cent. polynuclear. The Shick test was negative. Von Pirquet was negative. Cultures from the nose and throat were negative. The child looked well, was taking its food well and weighs the same now as on admission.

The disease from which this child was suffering is thus a matter of speculation. The physical signs would indicate a pneumonia while the X-ray picture would mean either a pneumonia or a sacculated empyema. Either diagnosis of pneumonia or empyema has this slight leucocytosis showing in the blood, the very low polynuclear, and the complete absence of fever. Moreover, a needle of large calibre inserted into this region from the axilla showed no fluid.

One other lesion might be the cause of these signs and the shadow in the X-ray plate, and that is tuberculosis. Against this, we have an X-ray picture that is not at all typical of tuberculosis, and physical signs that are better defined than would ordinarily be found over such an area of tuberculosis.

During the twelve days the child has been in the hospital, there has been practically no change in either the physical signs or the X-ray picture.

#### ACUTE MILIARY TUBERCULOSIS

This child, twelve months old, was admitted to the hospital six weeks ago, with a history of having had whooping cough for the previous six weeks. He is said to have lost weight, and three days ago developed a fever without chill or convulsions. Yesterday, his fever was reduced by active catharsis, with no marked cough, and a temperature of 103° F.

No history of previous illness.

On admission, blood examination showed about 12,500 white blood-cells and 61 per cent. polynuclear. Positive Von Pirquet and positive Shick; nose and throat cultures were Klebs Loeffler. Recently, the temperature has not gone above 103 and has shown less marked remissions.

On physical examination, the baby was well developed, well nour-

ished, with flushed cheek and sweating skin. He showed mild dyspnoea. Pharynx was somewhat red. No teeth present. No enlarged cervical lymph-nodes. Examination of the chest, showed only occasional râles and no evidence of localized lesion. X-ray plate of this child showed many tubercles throughout both lungs and a small area of consolidation in the left axillary region.

The spleen is palpable  $3/4''$  below the free border of the ribs. On the basis of the physical examination, and the X-ray plate, a provisional diagnosis of general miliary tuberculosis was made, and the child being less than a year old, with evidence already of involvement of the spleen, a very bad prognosis was made.

During the six weeks that the child has been here, he has been kept on the roof and as far as possible in the sun. The boy is not only receiving fresh air for twenty-four hours but on each fine day, is exposed naked to the sun. As soon as the skin becomes used to the direct rays of the sun so that he does not burn, he will remain naked in the sun most of each fine day. The child has been running an intermittent temperature, with marked remissions, going as high as 104 and as low as 98, without physical signs in the chest, and with repeated X-ray plates showing for some time rather increased lung involvement, but it seems possible in this case, in spite of the considerable spread of the lesion and the early age of the child, that a limitation of the spread of the lesion and possible healing may be obtained. He has had no medicine except rhubarb and soda.

The boy has lost less than 2 pounds since admission and the recent X-ray plates show no increase in the number of tubercles present. On this account, we have given more active treatment, which in older children with tuberculosis of the lungs, has been found moderately curable.

#### ACUTE DILATATION OF THE HEART

This girl, aged eleven years, was admitted ten days ago. She complained of a swelling in the mid-epigastric region which first appeared 8 days ago, at which time she went to a doctor, and he told her her heart was affected. The pain was worse after eating, and four days ago the lump disappeared. She complains of dyspnoea on exertion and sweating at night. Had emaciated during the last month.

At times, she has had slight pericardial pain. She says a few weeks ago, she had pains in her right ankle, and that there was a swelling of the foot and ankle which lasted three days.

On admission, her temperature was 102.5 and the blood showed 80 per cent. hæmoglobin, 8600 white cells, and 85 per cent. polynuclear. The Shick and Von Pirquet tests and throat and nose cultures were negative.

Examination of the heart showed a very indefinite apex impulse, and the left border of the heart could not be well mapped out. Coarse pericardial friction rubs could be heard over the whole pericardium and at that time, no murmur was heard.

The X-ray picture of the heart showed a very large heart, extending to the chest wall on the left side, the shadow being rather thin toward the axillary line. There was no elimination of the cardiophrenic angle, indicating that no fluid had been secreted in the pericardial sac.

The following day, the pericardial signs were still present. The third day, they had diminished considerably, and now a systolic blowing murmur could be heard at the apex. On the fifth day after admission, the pericardial signs and the murmur had entirely diminished, and the child's general condition considerably improved.

On the fifth day, the temperature reached normal and has not been above normal since. The second X-ray picture taken six days after admission shows a heart of nearly normal dimensions, clear outline, transverse diameter being  $4\frac{3}{4}$ " as against 6" in the first picture taken.

At the present time, this girl is apparently well. She has no physical indications of any lesion. This case is apparently a case of acute dilatation of the heart, probably following a rheumatic infection and due probably to acute digestive disturbances together with over-exertion. Under good conditions, she has very promptly recovered from what was apparently a very serious disorder.

# **Industrial Medicine**

---

## **INDUSTRIAL SURGICAL CLINICS**

**By PAUL B. MAGNUSON, M.D.**

Attending Surgeon Wesley Memorial Hospital and Alexian Brothers Hospital,  
Instructor in Surgery Northwestern University Medical School, Chicago,  
Consulting Surgeon O'Gara Coal Company. Formerly Medical Director,  
Illinois Industrial Commission; Chief Surgeon, Chicago and Alton  
Railroad, Chicago Junction Railroad, and Union  
Stock Yards and Transit Company

**AND**

**JOHN S. COULTER, M.D., F.A.C.S.**

Formerly Lt. Colonel, Medical Corps, U. S. Army. Consulting Surgeon  
Nokomis Coal Company

---

## **PHYSIOTHERAPY IN INDUSTRIAL SURGERY**

PHYSIOTHERAPY proved its value during the war in the treatment of war injuries, and is now proving its value in the treatment of practically the same class of injuries in industry. Physiotherapy includes treatment with heat, massage, electricity, exercises, water and light.

Physiotherapy according to its time of use may be classified as—

(1) Preventive, which is not used in injuries as much as it should be. It is all too frequent for us to see cases of fractures many weeks after the injury, in which the disability is not from the fracture, but from stiff joints due to lack of use. The patients all tell the same story: "I wore a splint for six weeks, then the doctor told me to use my arm (or leg) at home." Unfortunately, after six weeks immobilization a joint is stiff, use causes pain and the pain prevents the patient from using it. It should be recognized that skilled physiotherapy can be given at a far earlier period than is usually allowed. During the war we learned that in infections preventive physiotherapy saved many marked disabilities.

(2) Corrective or curative physiotherapy is that which is used to correct or cure disabilities following injuries, and is, of course,

closely associated with the preventive. The more preventive physiotherapy is used, the less corrective is necessary. To correct or cure a disability from an injury is a more difficult task and takes much more time than to prevent the same disability.

#### CLASSES OF INJURIES NEEDING PHYSIOTHERAPY

(1) *Sprains*.—Sprained joints, if placed at rest and lotions used, usually owe their subsequent stiffness to the treatment, not to the injury.

To restore a sprained joint to normal motion we use diathermy ten to thirty minutes; massage which should be derivative and not local, and early motion. This motion should be passive and limited by the onset of pain. If this rule is observed no damage is done to any torn ligaments.

(2) *Dislocations*.—Massage and exercises should be started at once after reduction, under the care of the surgeon. In fact, all joint motions in a recently reduced dislocation should be done by the surgeon himself after baking and massage of the injured joint. The greatest care is essential lest the dislocation recur; therefore, motion should be gradual and not at too great an angle before a week or ten days have passed.

The function of a joint is motion and a joint not moved will become stiff, also the muscles moving a joint are usually supplied by the same nerves as the joint itself, and there is a reflex atrophy of these muscles. This can only be prevented by massage, electric stimulation and proper motion. This is especially important in the shoulder joint as the stability of that joint is dependent upon the surrounding muscle tone.

One case we recently saw on examination had a posterior dislocation of the elbow joint, properly reduced, and then treated with his elbow flexed, fingers extended and bound to his side for four weeks. Then he was given a sling and told to use it. It pained on extension and his fingers hurt on flexion, so four weeks later, he came to our clinic, and it took three months of daily physiotherapy to restore him to within 25 per cent. of normal motion in the elbow, wrist and fingers.

(3) *Ankylosis of Joints*.—Most of the industrial cases in which this form of treatment is now used, are those having stiff joints. These cases in most instances would have been avoided by proper



physiotherapy during the treatment for the original injury, using the same means now used to cure the stiffness. In other words, the insurance companies and employers are now paying a high cost for this treatment as a curative and corrective measure, when they should have included in the original treatment cost the same measure used as a preventive treatment against stiff joints. To pay a surgeon the cost of a visit to a fracture or a dislocation case in which he only looks at the splint, is not only a waste of money, but is eventually going to cost many more visits of someone skilled in physiotherapy to cure the stiff joints following prolonged immobilization. It is not supposed that the surgeon will give massage, but he should see that it is given and he should either give or supervise all joint motions in recent cases.

In all treatment of stiff joints, cases of bony ankylosis and those cases where there is some contra-indication by a pathological process, such as tuberculosis, should be excluded. Where there is a bony obstacle it is useless without surgery to attempt to gain motion beyond the bony blocking. So we have to treat here by physiotherapy, stiffness caused by adhesions inside or outside the joint, by inflammatory changes or by retraction of scar tissue.

In our clinic for the treatment of stiff joints and the prevention of stiff joints in closed cases, we use in addition to baking, massage and hydrotherapy, a negative galvanic current with a clay electrode.

*Fractures* are now usually treated by one of three methods:

(1) Complete immobilization, until fragments are united, by splints or plaster casts.

(2) Relative immobilization by traction, best seen with the Thomas splint and traction.

(3) With physiotherapy added to one of the above.

By far the most fractures that we have sent to us for corrective treatment come under the first class.

When a bone is broken the surrounding structures are injured also. Rigid fixation by splints or plaster without physiotherapy withholds from these structures the treatment by which repair can be accomplished, and thus we have added to the original injury, adhesions and atrophy of disuse.

Massage and indirect diathermy in the first stage of a fracture hastens the absorption of the hematoma and injured tissue cells, and

improves the metabolism of the injured parts. Thus it diminishes the pain and lessens the muscle contraction and consequently aids in the reduction and retention of the fragments. Fixation and rest diminish the blood supply, hence do not aid in the above.

Joint motion in a limb where there is a fracture prevents joint stiffness and muscle atrophy. This mobilization does not tend to disturb the alignment of the fragments, unless the conditions are such that an open operation is necessary to restore the fragments to proper position.

With massage, electrical muscle stimulation and joint motion, muscle atrophy and joint stiffness are prevented and the limb is ready for use when there is union. With complete immobilization there is marked muscle atrophy and joint stiffness and we get the result of a good union but a marked permanent disability from stiff joints.

There is one great requirement for this form of treatment for fracture cases—skill. If it takes a skilled surgeon to perform an open operation for an ununited fracture, so the surgeon who treats fractures by mobilization must be experienced and skilled.

We have recently seen two cases which will illustrate the above principles.

Case C. W., injured November 17, having a fracture of the surgical neck of the right humerus. He was treated by his family doctor, a general practitioner, with his arm bound to his chest, elbow flexed, fingers extended for four weeks, then in a sling for the three weeks following. January 28, he was sent to our clinic for corrective physiotherapy. He had an excellent union of his fracture, but an almost total loss of use of his arm. In his shoulder joint abduction and flexion were possible only 20 degrees, right elbow showed only 50 per cent. motion, fingers practically no flexion.

Three months' treatment was necessary at a cost of \$150. After he was discharged from his original treatment we estimated he had 80 per cent. loss of use of his arm, and after three months of daily physiotherapy treatment we were able to reduce this to 25 per cent. loss of use. At least half of this was from loss of motion in the elbow, wrist and fingers, which were never injured. Thus the original treatment added disability to other parts while curing the injury.

Case J.M., injured June 29, with exactly the same injury to his

right humerus, but added to this was a right Colle's fracture. He was treated from the start at our clinic by early massage, mobilization and electric stimulation of his muscles with the result that he returned to work on August 31, with only a 5 per cent. total permanent loss of use of his arm.

This man was sent to work, and did his normal work as a roofer, two months after his injury, which was about two weeks before case C.W. started his massage. He had the same injury plus another in the same arm, yet he returned to work three and a half months before the other with a 20 per cent. less permanent disability and with the cost of only the original treatment, because his arm was daily taken from the splint, massaged by an experienced masseuse, and careful joint motions given by the surgeon.

*Peripheral Nerve Injuries.*—Corrective splinting and physiotherapy are absolutely essential in the treatment of these conditions. As long as there are progressive signs of nerve regeneration no surgical interference is permissible, when these have ceased to advance or have not appeared, operation is indicated. In the after treatment of nerve injuries whether operated or not, the essentials are (1) physiological rest in splints to completely relax the paralyzed muscles and to prevent the active muscles from contracting; (2) restoration of function by physiotherapy.

The health of the motor muscles and nerves depends on use and degeneration follows disuse. The repair is a nutritional problem; therefore, electric stimulation and hyperemia are essential for restoration of function.

In our clinic we have these cases report for daily treatment, giving them baking, massage, joint movements, and slow sinusoidal electric stimulation. If the patient is from out of town and cannot come daily over the long period necessary for the treatment of these injuries, we teach him or a member of his family the use of the Bristow Coil (see issue *International Clinics*, vol. ii, series 30, June, 1920, Fig. 10-a). He uses this at home and reports twice monthly. In the previous issue we have given our treatment in detail with an illustrative case (see page 21, Case H.M.).

*Muscle and Tendon Injuries.*—In the after treatment of muscle and tendon sutures movement should always be started in the direction that relaxes the sutured tendon, and thus not put any strain on

the suture line. To turn over one of these cases to one who has had no special training is merely to court disaster. This movement should be started not later than three days after the operation, thereby preventing adhesions.

*Amputations.*—In the after treatment of amputation stumps the whirlpool bath was found to give the most satisfaction. These baths give a continuous bath of high temperature with a strong water current.

*Malingering.*—If these cases are given daily treatments, and consistent daily notes kept on their complaints and accurate observations made on their localization of tenderness, they will be shown to be malingering in a week's time, as their attention is distracted by the treatment and they will fail to correctly localize their supposed pain and tenderness.

#### COST

Employers and insurance companies are in business to make money, consequently are interested in the problems of making and saving money.

For the employer we have found that this treatment pays for itself and saves him money in compensation costs by reducing the period of temporary disability and the amount of permanent disability from injuries. We have given in these clinics many examples of this.

For the patient it saves him money by returning him to work sooner, and by reducing his permanent handicap from any disability. It also increases the morale of the patient, and keeps him satisfied, as he is having something done for him every day. He is not allowed to wear a splint for weeks, then have it removed and told to go use the injured part.

Several large industries have established their own departments to give physiotherapy, and it has in all cases resulted in a saving as just described. In view of this we believe that the organization of such a department would have a vital interest to industrial surgeons.

#### SUPERVISION

This department should be supervised by an experienced orthopedic surgeon, who has had training in this branch of work. Only too often when cases are sent or go to a masseur we see treatment well given, but other lines of treatment neglected. For instance,

we have seen cases of wrist drop from musculo-spiral paralysis given daily massage but allowed to go for the remainder of the day without a splint. Consequently for the lack of a "cock-up" splint, the extensors were permanently damaged by over extension and the flexors allowed to contract. In all cases of fracture, early motion can only be done properly by an experienced surgeon.

If the surgeon in charge of the physiotherapy is not the surgeon who operates or supervises the other treatment, there should be frequent consultations between the two, as often physiotherapy can be materially aided by the use of certain splints or other apparatus.

*Technicians.*—Physiotherapists are of three classes:

(1) Nurses who have had general training in hospital wards and then a post-graduate course in physiotherapy.

(2) Physical training teachers who have had courses in physical training and afterward a course in physiotherapy.

(3) Those who have had neither nursing nor physical education but who were trained as reconstruction aides during the war.

Of the three the nurse should make the most valuable administrator of this form of treatment, as (1) she has had experience in the importance of the execution of the doctor's orders; (2) she has had a general experience in the handling of injuries; (3) she has had surgical nursing training and is able to do many of the dressings in a correct manner.

*Pay.*—In the U. S. Public Health Service they pay their female reconstruction aides \$80 per month, plus board, room and laundry. In the offices we have established in the coal mining fields we have paid \$100 per month, plus room, board and laundry. A nurse with physiotherapy training is usually paid \$200 per month, without board, room or laundry.

*Number of Cases.*—A single technician can handle from sixteen to twenty industrial injury cases a day, if these are carefully arranged as to time and the kind of treatment needed in each case.

#### EQUIPMENT

The authors have established two physiotherapy departments for the after treatment of cases injured in coal mines. One was at Nokomis, Illinois, and the other at Harrisburg, Illinois.

The one at Nokomis was the first one established in the mining industry of Illinois and was authorized by the Indiana and Illinois

Coal Corporation, T. C. Keller, President. The space used was vacant space in their local office, and consisted of two steam-heated rooms, one for treatment and one for a waiting room. It was established as an experiment at a minimum expense, and consisted of the following equipment:

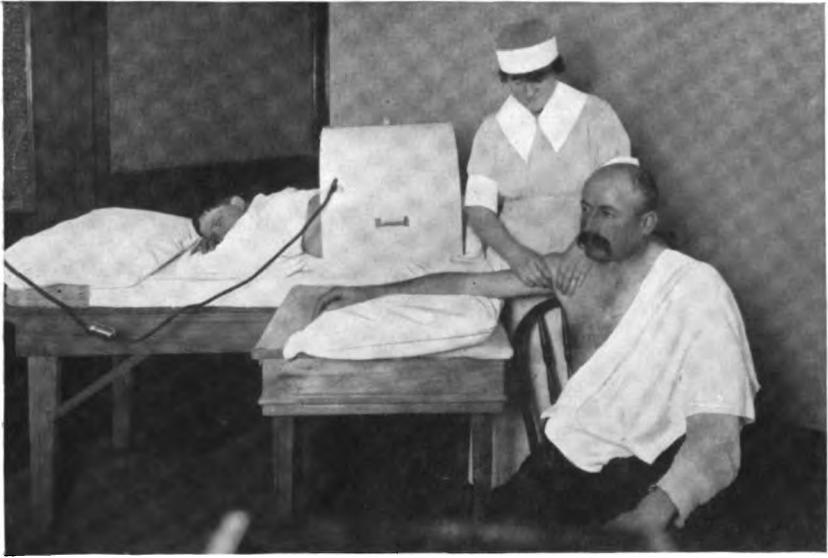
1 Baker "Burdick" type B-2 .....	\$ 75.00
1 High-frequency Wappler Telatherm Jr. ..	150.00
2 Mattresses .....	25.00
2 Tables .....	20.00
4 Pillows .....	12.00
2 Blankets .....	10.00
1 Doz. sheets .....	24.00
2 Doz. pillow cases .....	14.00
1 Doz. bath towels .....	6.00
1 Doz. hand towels .....	3.00
6 Pr. pajamas .....	24.00
3 Bathrobes .....	15.00
3 Chairs .....	15.00
Supplies, powder, cocoa butter, dressings, etc. ....	7.00
	<hr/>
	\$ 400.00

This office with one girl and two tables treated from sixteen to eighteen patients daily. On the first visit as consulting surgeon to Nokomis, it was found that there were fifteen cases that absolutely needed this form of treatment for their recovery. It was calculated that to send these cases to the city for this treatment would cost about \$4 a case per day, or about \$1800 a month for all. So with an original expenditure of \$400 and a monthly expense of less than \$200, these cases were treated at their homes. (See Fig. 1 and 2.)

#### EQUIPMENT OF AN OFFICE TO TREAT FORTY PATIENTS DAILY

To treat forty patients daily we believe that about 600 square feet of floor space is necessary, divided into four treatment rooms, examination, waiting and dressing rooms. The partitions can be sheets on wires, wood or wood and glass. At the doors are brown burlap curtains. The arrangement of the four treatment rooms with the doors all opening at one centre is believed superior to the arrangement of the rooms in a row, as it gives the technicians less distance to travel and gives better control of the patients, so that a patient can be left under a baker while the nurse is giving massage or electricity to another case.

FIG. 1.



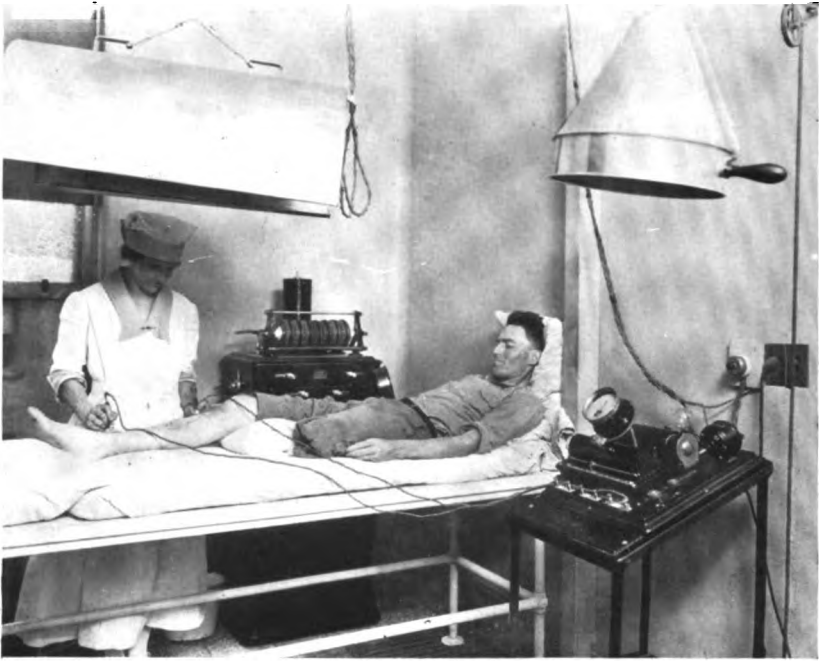
Physiotherapy in Nokomis, Ill., coal mining town. Treating two patients at once—the man under the baker is a sacro-iliac subluxation case and the one receiving massage is a case of subdeltoid bursitis.

FIG. 2.



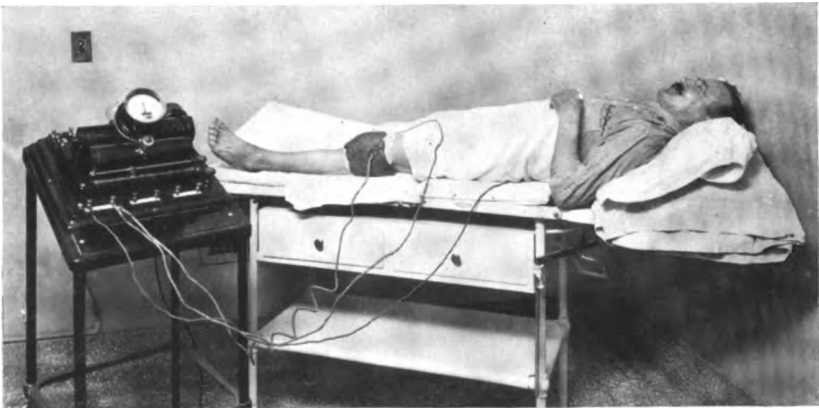
Physiotherapy in the coal mining industry—another view of the office at Nokomis showing another method of treating two patients at the same time, on the table is a case of dislocation left hip joint.

FIG. 3.



Physiotherapy Laboratory at the Illinois Steel Corporation, Gary, Ind., shows method of distribution of apparatus so that patient can be given light or heat treatment or massaged; then given electric stimulation without moving patient or apparatus. The baker and the deep therapy light are suspended from the ceiling by ropes and pulleys.

FIG. 4.



Physiotherapy in the Steel Industry, at Illinois Steel Corp., Gary, Ind., showing treatment by galvanic current with clay electrodes for a stiff knee. Notice patient now does not require the services of the technician who is treating another case. She will return to this case and give massage and joint movements.



The treatment tables are made by a local carpenter and stained with mahogany stain. These measure: Top, 6 ft. 2 in. x 2 ft. 6 in.; height, 2 ft. 8 in.

In this plan each room has clothes hooks and the patients dress in the room.

*Waiting Room*

1 Table .....	\$	8.00
6 Chairs .....		20.00

*Dressing Room*

(Painted white, with stationary washstand)

1 Small electric sterilizer .....	\$	32.00
1 White iron instrument table .....		15.00
1 Steel and glass instrument cabinet .....		100.00
(To hold instruments and dressings)		
2 White chairs .....		10.00
1 Air cooled ultra-violet lamp Burdick ....		425.00
(to aid in treating superficial infections)		

*Treatment Rooms*

4 Tables .....		40.00
4 Mattresses .....		20.00
6 Pillows .....		6.00
12 Sheets .....		12.00
2 Tables (small hand cases) .....		10.00
6 Chairs .....		20.00
6 Blankets .....		15.00
12 Pillow cases .....		5.00
1 Burdick baker (large) .....		75.00
1 Burdick baker (small) .....		38.00
1 Combistat victor for alternating current.		640.00
Accessory electrodes, etc. ....		35.00
Galvanic control with 1-ampere motor-generator .....		280.00
(Above three will give all low tension modalities)		
High-frequency apparatus model Wantz ..		650.00
Hot wire metre .....		35.00
Spray electrode .....		2.50
2 Surface non-vacuum electrodes .....		15.00
Total .....		\$2508.50

This is practically the equipment used in the O'Gara Coal Company's new physiotherapy laboratory at Harrisburg, Illinois, and one at the Gary Hospital of the Illinois Steel Corporation (see Figures 3, 4 and 5).

# LAMINECTOMY FOR FRACTURES OF THE LAMINÆ OF THE TWELFTH DORSAL, FIRST, SECOND AND THIRD LUMBAR VERTEBRÆ

By WM. A. HENDRICKS, M.D.

L. B., age twenty-seven, male, white, electrical engineer. Admitted 5-21-21—Discharged 6-25-21.

*Chief Complaint.*—Incontinence of feces and urine, some retention of urine, numbness of both feet, loss of use of the legs, stiffness of the right wrist.

*History of Present Illness.*—Four weeks ago patient fell fourteen feet, striking across the sacral region while working on a ladder doing some wiring. He was taken home where he remained for three days and then sent to a hospital. He had loss of function in the lower limbs. He had areas of anaesthesia over the region of the peroneal nerves, over the dorsum and plantar surfaces of the feet; he had some numbness in both feet and he had an injured right wrist. After he was in the hospital about three weeks, consultation was asked and conclusion arrived at that this man had a fracture of some lumbar or dorsal vertebra. He required catheterization daily and had incontinence of bowels.

X-ray pictures were taken which showed a luxation of the scaphoid and fractures of the laminæ of the twelfth dorsal, first, second and third lumbar. The wrist at this time is limited in motion, swelling has subsided.

He was transferred to another hospital for surgery. It is hoped that relief of pressure on the cord will minimize the possibility of a total and permanent loss of use of the lower limb and markedly improve, if not entirely clear up, the bladder and rectum symptoms.

*Gastro-intestinal System.*—He gives a history of belching of gas and some sour eructations, and at the present time, incontinence of feces, but at no time, either at the time of the accident or now is there any history of passing blood by bowels.

*Cardio-vascular System.*—No dyspnoea, no vertigo, no oedema, no palpitation, no precordial pain.

*Pulmonary System.*—Has occasionally been subject to colds and several years ago had a persistent cough. He weighs about 137 pounds, being 5' 11" tall. Has had no sudden loss of weight. Gives a history of night sweats; no history of spitting of blood.

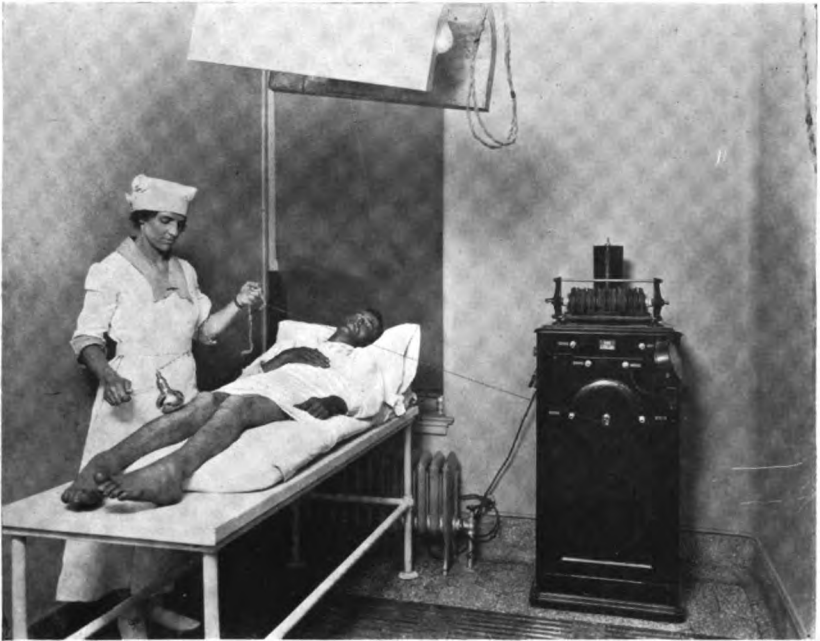
*Genito-urinary System.*—No frequency previous to the injury, no burning, no passing of blood, neither at the time of the injury nor now. He now has retension with some incontinence with an evidence of a cystitis.

*Nervous System.*—Seems quite nervous. Now sleeps irregularly, eats poorly. Has a loss of nerve sensation, distribution over both lower limbs extending over the lateral thigh to the front of the knee laterally and posterior lower leg and foot.

*Past Medical History.*—Has had measles, mumps and whooping cough; no typhoid, no scarlet fever, no rheumatism; has had no previous accidents and no previous operations.

*Family History.*—Father and mother living and well. Wife living and well; no children. No family history of tuberculosis, cancer, heart, or kidney disease, diabetes, rheumatism or epilepsy.

**FIG. 5.**



**Physiotherapy at Illinois Steel Corp., Gary, Ind., showing another room. Technician is treating a case of spastic paralysis from a spinal injury, note baker is pulled up after giving treatment.**



*Physical Examination.*—Patient is an adult male, tall and rangy type with some presence of noticeable loss of weight at this time. He is pale, nervous and restless. On gross inspection, it is noted he has lost use of his lower limbs. There is over the region of the sacrum about the size of a silver dollar, a pressure sore. Blood pressure is 122 systolic and 88 diastolic; pulse, 72; temperature, 97; respirations, 14.

Head—Scalp negative.

Eyes—Pupils enlarged, react to light and to accommodation; sclera clear.

Nose—Negative.

Ears—Negative.

Mouth—He has some evidence of cavitation; no pyorrhea. Tongue is coated, moist, slight tremor. Mucous membrane is moist. In the region of the tonsil posterior pharynx is injected; not cryptic.

Neck—Long, scrawny neck, some visible pulsation of the large vessels. Submaxillary glands enlarged. Post cervicals enlarged; thyroid not palpable, not markedly enlarged. Subclavicular very prominent depression.

#### CHEST

*Lungs.*—Inspection—Chest is long and flat. Expansion is fairly good; slightly sluggish on the right side at the apex. No areas of tumor masses; infra-clavicular space is prominent in its depression.

Palpation—Expansion is fair. Vocal fremitus normal; some slight decrease in fremitus at the right side posteriorly.

Percussion—Impaired note at right apex posteriorly. No dullness or flatness otherwise. Outline of lung normal.

Auscultation—Increase in voice sound at the right apex and some increased whispered voice conduction; expiration prolonged at the right apex, some musical râles in this neighborhood.

*Heart.*—Inspection—Apex beat in the fifth interspace at the nipple line. No enlargement, no precordial bulging or pulsation.

Palpation—There is felt no thrills; heart beat is regular and full.

Percussion—Heart outline is normal.

Auscultation—There are no murmurs. Heart rate is regular and muscle tone is of good quality.

## ABDOMEN

**Inspection**—Scaphoid; there is no visible peristalsis.

**Palpation**—No areas of tenderness, no tumor masses, no ptosis of the abdominal viscera.

**Percussion**—The outline of the abdominal viscera normal; no dullness; tympanitic note throughout.

**Auscultation**—Peristalsis normal.

## EXTREMITIES

Loss of function of the lower limb; neurological examination reads as follows: Paralysis of all muscles supplied by the sciatics, both sides, excepting the hamstring muscles which are slightly weaker on the right side. Generalized weakness of the thigh muscles. Paralysis of extensor digitorum communis, abductor pollicis and extensor pollicis longis of the left hand. Reflexes: Achilles jerks absent; knee jerk normal on right, slightly greater than left. Plantars absent. Cremasteric brisk right, greater than left. Abdominalis right normal, left diminished. Arm jerks brisk, except the triceps which are diminished, marked atrophy of tibialis group on both sides. Analgesia over the fifth lumbar, first, second, third, fourth, fifth and sixth sacral segments. Incontinence of feces, but is beginning to feel movement and can feel sensation of urination; can express urine with the aid of a gag reflex. No analgesia over the musculo-spiral nerve on the left side.

## LABORATORY

**Blood Analysis**.—Red cells, 4,967,000; 10,600 whites; 90 per cent. hæmoglobin. Blood clotting time, four minutes.

**Urine**.—Color, amber; much sediment; much albumin; S. G., 1024; reaction, alkali; sugar, none; no casts. R.B.C., many; W.B.C., many. Urine was obtained by catheterization.

**Operation**.—It was decided that laminectomy be done for the relief of pressure on the cord.

Incision in the upper lumbar and lower dorsal through the fascia of the erector spinæ muscles to the spinous process was made. The heavy muscles of the back were dissected free from the lamina. Spinous processes of the twelfth dorsal, first, second and third lumbar were entirely removed. The laminæ of the bodies of the first and second lumbar removed, exposing the spinal cord. Few fibrous

bands divided, freeing the cord. Dura not opened. There is seen a duplicate kinking of the spinal cord at the first lumbar vertebra, resembling a double camel's hump. Few bands of fibrous tissue restricting cord below this level. No tumor mass found. Canal probed distally, found to be patulous; rubber dam drain in the lower angle of the incision; catgut used to suture the fascia and muscle; interrupted silkworm gut used to suture skin. Heavy cotton pads placed over the back. Patient removed to the room.

Four days following operation the temperature was 102.4; pulse, 100; respirations, 20. It is difficult to keep the wound of the back dry and to care for the pressure sores which are now increasing in size because of the moisture of the incontinence.

Eight days after the operation, bed sores are increasing in size. It is attempted to seal them up with rubber dam dressing. The man still has incontinence, but has a sensation of urination. No marked increase of nerve or muscle strength. Galvanism tried for both legs, sufficient to produce good contraction. Faradic current to extensors of fingers and thumb. Patient eats poorly. Says he can void some if he gags himself and this he does by placing his finger or the smooth end of a tooth brush down his throat.

Thirteen days after operation the temperature is 100.6; pulse, 100; respirations, 18. Stitches have been removed and the back wound is clean and dry, but the bed sores are very slowly healing in from the edges, partly because of the position in bed and partly because of moisture. Patient's appetite is poor. Sleeps poorly, is losing some weight. An air mattress is obtained for the comfort of the patient and also to attempt to limit the bed sores.

Twenty-four days after operation he is receiving daily electrical treatments and requires catheterization. Appetite is poor. Temperature on this date is 101; pulse, 100; respiration, 20. Patient is gotten up in wheel chair in a reclining position and has a sensation of fainting and feels weak on attempting to remain there. He is removed to bed. The back wound is clean. Bed sores over the sacral region still present and showing little sign of tendency to heal. Patient eats poorly; is given all the nourishment of soft and liquid diet, and some stimulations, that it is possible for him to take. Complains of soreness of throat. Spits up a phlegm. Sputum examination for tuberculosis is negative.

Twenty-seven days after operation temperature is 102; respirations, 24. It is noted this patient is fast slipping down hill; he cannot be induced to eat. Complains of no discomfort or pain. Still has involuntary bowel movements. The bed sore in the sacral region is very slowly filling in but there is still some depth and drainage. It was decided at a conference with the family and physician that it may be just as well for the patient to be moved home, inasmuch as he is not improving. In fact, he is losing weight rapidly, sleeping poorly and eating poorly. Possibly by more detailed attention at home he could be encouraged to take more food, although it is well known that the prognosis is poor; the kidney function being markedly impaired, having for a long time a septic temperature and no signs of improvement of the paralyzed areas.

After being at home four days, the patient having persistently lost ground, died on the thirty-seventh day following operation.

An autopsy revealed death was due to bilateral pyonephrosis; the bladder showed some slight damage of the mucosa and some thickening of the bladder wall; a fatty infiltration of the cardiac muscle and a fatty infiltration of the liver; both the cardiac and liver circumstances being due probably to the generalized septic disturbance. Right apex an infiltration the size of a walnut well defined, some slight caseation.

*Comments.*—It is to be noted that this man was in the hospital with this injury for about three weeks before consultation was requested for operative procedure. This, in spite of the fact that there was a fracture of the laminae of the twelfth dorsal, first, second and third lumbar vertebrae shown in the X-ray, with the loss of function in the lower limbs and incontinence of feces and urine. It is believed that a laminectomy should have been done in this case, during the first week, as it was evident from the X-ray and examinations that there was a cord compression due to the injury and this was proved to be the case by the operation.

There were no symptoms in this case indicating a gradual loss of sensation or a gradual paralysis of the muscles, which would indicate that the compression was due to a hemorrhage.

Fraser has summarized the indications for operation in spinal injuries as follows:

In dislocations, provided the facilities for performing a laminectomy



tomy are available, the open method of reducing dislocations is safer than the closed.

Fractures of the laminae with cord or spinal root lesions and no evidence of spontaneous improvement within the first week should be operated.

In fractures of the spinous or transverse processes when there is continuous pain and disability, suggest the presence of an ununited fracture or exuberent callus and should be operated.

Fracture dislocations, even though in the majority of cases, the cord is irreparably damaged at the time of accident, there is a small chance that immediate operation may relieve an acute compression at the margin of the fragment.

It is to be noted that this case showed the usual consequences of a spinal cord injury and that there were marked bed sores and a marked bladder infection; the bed sores eventually were nearly healed, due to very careful nursing attention and the use of an air mattress. The bladder infection went on to a pyelitis and a pyonephrosis; and this pyonephrosis, the post-mortem revealed, was the cause of his death.

---

## PNEUMONOCONIOSIS

J. W., age fifty-two. Occupation, sand-blast worker.

*History of Present Condition.*—This man states that four months ago he first noticed his present trouble and that it started with pain in the upper part of his chest, and he noticed that after working a while he had a slight cough. This cough grew progressively worse, so that two months thereafter he had to stop working on account of the severe coughing whenever he exercised or even walked fast. He had no pain in the upper part of his chest. This coughing came on in paroxysms and the sputum was thin, serous and streaked with black. He had always slept well and can sleep well at this time. Four months ago he weighed 178 pounds; he now weighs 162 pounds.

*Previous Medical History.*—He had a foot infection twenty-five years ago; he had some cough about two years ago, but this cleared up. He had acute gonorrheal urethritis thirty-five years ago.

*Family History.*—Mother died of tuberculosis. Father of old age at 82. He has three children living and well; four children died in infancy.

*Physical Examination.*—The nose shows a congenital deformity with the nasal septum deflected to the right.

Teeth are in fair condition; about six are missing and six others filled.

Tonsils are not enlarged. There is slight pharyngitis; sub-maxillary lymph glands are enlarged.

The right lung from the apex to the third rib anteriorly on percussion is slightly dull; palpation, increased tactile fremitus; auscultation shows increased voice sounds and marked bronchial breathing. At one point in this area just to the right of the sternum, there are signs that would almost indicate a cavity, but on percussion the area was dull. There were a number of râles over this whole area and over the left apex. The remaining areas of the chest were negative.

The heart dullness was normal in outline except that it extended upward to the top of the sternum. There were no murmurs present.

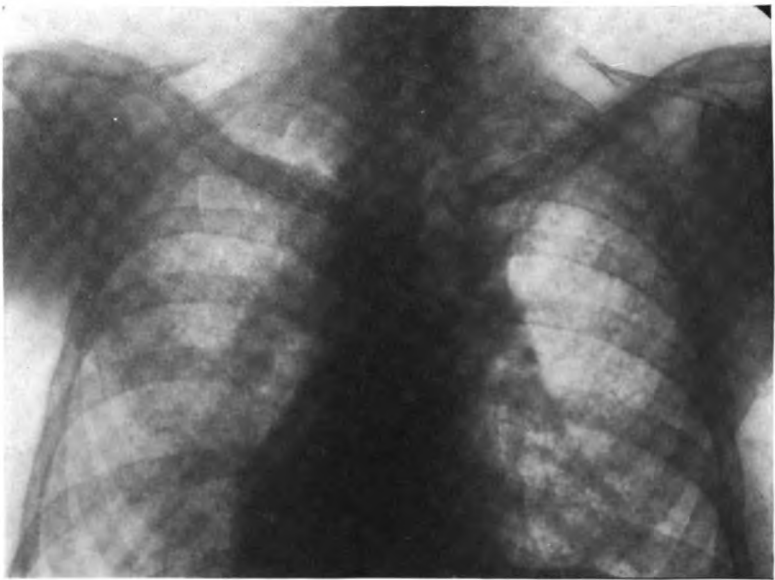
Examination of the abdomen: Both inguinal canals were filled with a hard substance which on questioning the patient was found to be paraffin which was injected for the cure of hernia some years ago. There was a large inguinal hernia on the right side. There was no evidence of venereal disease. At no time had this man any temperature.

*Laboratory Examination.*—Sputum examination was negative for tuberculosis.

*X-ray* by Dr. Hollis E. Potter showed the following (see Figure 6): Throughout the lung fields, there are small scar consolidations of the type often seen after the inhalation of dust. There is much increase in the tissue at the hilus of either side in the upper mediastinum. The trachea is pulled decidedly to the right, there being no evidence of compression of the air column. This is associated with a fibrosis and calcification of the right apex, and is probably due to traction after scar formation in this region. There is no reason to believe that the infiltration of the upper mediastinum is of any different character than is seen in the lungs. There is no evidence of aneurysm or other changes in the circulatory organs.

*Comments.*—This man came before our clinic for examination to determine whether he was injured, as he claimed his whole trouble started from a definite date, after some unusually heavy work. It was decided after the above history and examination that he was suffering from pneumoconiosis, due to working on a sand-blast machine and inhaling small particles of sand, which have

FIG. 6.



Pneumoconiosis: X-ray by Dr. Hollis E. Potter showing a marked increase in the tissue at the hilus of either side in the upper mediastinum. Throughout the lung there are small consolidations of the type seen after the inhalation of dust. The trachea is pulled decidedly to the right with no evidence of compression. There is fibrosis and calcification in the right index.



infiltrated his lungs and his mediastinal glands, as is shown in the X-ray picture.

His cough is apparently caused by the irritation of the enlarged mediastinal glands and the pulling to the right of his trachea. He has not much bronchitis, and his cough is practically all an irritative one, as his sputum is slight in amount. His cough comes on only after exercise; he can sleep well and it does not trouble him except when he exercises which causes his lungs to expand more rapidly than normal.

The most recent literature on this subject is an article by Winslow and Greenberg in the *Journal of Industrial Hygiene*, January and February, 1921, on industrial tuberculosis and the control of factory dust problem. In this article there are given some very interesting statistics and a table of determinations of dust contents of air in the various types of industrial establishments. They find that the sand-blast chamber contains the highest rate of dust in air of any industry, giving in ten samples an average of 55.29 milligrams of dust per cubic foot, but the actual number of small particles present is less than in the air of abrasive factories, being 60,880,000 cubic feet against 159,779,000.

They also discuss the control of the dust, by four methods:

- (1) Substitution of wet for dry processes.
- (2) By conducting these processes in enclosed chambers.
- (3) By operation of hoods and the use of local exhaust ventilation.
- (4) By wearing of respirators and helmets.

The man is provided of course, with a natural means of protecting himself against dust inhalation and the nose is especially an efficient dust filter, and much of the dust is arrested in the nose and the posterior wall of the pharynx. Therefore it is common to find, in cases suffering from the symptoms of dust inhalation that there is some nose condition causing the man to be a mouth breather rather than a nose breather. In this case it is to be noted that this man had a deflected septum and he is a mouth breather.

It is noted in an article by Hayhurst in the *American Journal of Public Health*, January, 1920, that the horse in coal mines has no pneumoconiosis, due to the fact that he is an exclusive nose breather.

X-rays in these cases are characteristic except perhaps in the early stages. The X-rays of a like condition have been studied and described by E. C. Jarvis in the *American Journal of Röntgenology*, in May, 1921. There are really three stages as seen in the X-ray picture. In the first stage there is shown an increase in the shadows at the hilus and a thickening of the trunk shadows. The second stage is shown by more or less uniformly mottling throughout the lung structure, due to the deposition of dust in the lymph space, cells and fibrous tissue, with a certain amount of localized fibrosis. The third stage is characterized by the diffused fibrosis; dense fibrous bands can be seen extending in various directions and in this case it is believed that one of these fibrous bands has pulled the trachea to the right. Sometimes these fibrous bands are attached to the diaphragm, causing marked retraction. The heart and the blood-vessels are sometimes dragged out of place. The only way in which fibrosis from tuberculosis and pneumoconiosis can be differentiated between is by repeated sputum examinations.

This whole subject from a pathological and clinical standpoint, has in our opinion, been best taken up in an article by Landis in the *Journal of Industrial Hygiene*, July, 1919. This article covers the whole subject and gives a complete bibliography up to date.

In our clinic we have seen many cases of injuries in miners and in some of these it was necessary to take an X-ray of the dorsal spine and other views that secure chest pictures, and we have many times noted advanced cases of pneumoconiosis without a symptom, and many of the observers of this condition have noted that individuals vary in their resistance to dust.

In the Illinois coal miners it was found that the percentage death rate for tuberculosis in one census was 14.6, as against 16.6 for occupied males of the same area. The authors are both constantly examining miners for injuries, and it has been our experience that tuberculosis is less prevalent in coal miners than in ordinary workers.

Wainwright and Nichols, in the *American Journal of Medical Science*, 1905, in their study, found that the coal miners were less susceptible to tuberculosis than other individuals, due to the diffuse fibroid changes in their lungs. The pathology of this condition has been well described by Landis in his article and he divides it into the following stages:

(1) The very earliest changes in which the alveolar epithelial cells have become enlarged and contain a few particles of dust, and dust particles are seen in the walls of the small bronchi. There is no evidence in this stage of connective tissue proliferation.

(2) The second stage, the dust particles have gained an entrance to the lymphatic channels and there are a number of large mononuclear phagocytes filled with black dust particles present. These are first mainly in the lymphoid tissue between the alveoli. There is a small amount of perivascular and peribronchial fibrosis.

(3) The above mentioned fibrosis becomes more marked. There is fibrosis around the alveoli and larger bronchi, and small nodular collections of fibrosis appear. In this stage there is extensive blocking and obliteration of the small and medium size lymphatics and the bronchial and mediastinal lymph-nodes are enlarged.

(4) The final and advanced stage is characterized by a general nodular dissemination throughout both lungs, the formation of large fibrous areas at times.

## HODGKIN'S DISEASE: CLAIMED INJURY

By Wm. A. Hendricks, M.D.

I. D., male, age thirty-four. Occupation, coal miner.

*History of Present Condition.*—In February, 1921, a load of coal fell on him and injured both his arms and lower ribs on the right side; he states also that his back was injured and that he is now unable to make good use of his left arm; particularly is it painful on lifting above a right angle. This arm is smaller and shows some atrophy of the shoulder muscles.

*Past Medical History.*—He has had the usual childhood diseases. He has had neither influenza nor pneumonia; has never had malaria. Denies any history of venereal disease. He has been out of work for about two and one-half to three months previous to coming to this hospital, having been under observation and treatment at another hospital for a period of three weeks.

*Physical Examination.*—Patient looks markedly emaciated and has a pasty complexion. Examination of his eyes shows that he has a tendency to a yellowish tingeing of the sclera. His pupils are normal in size and react to light and to accommodation. Examination of the nose and ears is negative. Examination of the mouth reveals a somewhat pale and anemic mucous membrane; tongue is coated and slightly discolored from tobacco. His teeth are unclean. There are no evidences of infection of the gums, no cavities nor remaining roots. Larynx and pharynx are negative. Examination of his neck shows a long, thin neck, showing signs of some emaciation and a mass of glands in the right clavicle region, the larger of which is the size of an olive; the smaller about the size of a small marble. The supra-clavicular space shows marked depression. There is no visible pulsation in the larger vessels.

The chest is long and flat. Expansion is equal and fair. Voice transmission is normal throughout the chest. There are no râles present on ordinary or deep breathing, nor are there any present after coughing. There is slight dullness over the right base and also in the substernal region.

The heart is normal in outline. There are no thrills palpable. Heart sounds are regular in rate and rhythm. Muscle tones are of good quality. There are no murmurs present.

Abdomen is negative for any palpable masses. Spleen and liver are normal in size.



Blood-pressure taken on admission was 108 systolic, 68 diastolic. About five days later blood-pressure was 118 systolic and 70 diastolic.

Generally, this individual looked like he might be a tubercular patient, which suspicion had also been aroused in the minds of investigators of this case in another hospital, several months past. He had on admission a temperature ranging between 98 and 101, at times reaching 101.8. The pulse rate ranged between 88 and 118.

*X-rays* were taken to investigate the condition of the chest, which showed a pronounced increase in the structure of the hilus, which could only be accounted for by an infiltration of the peribronchial glands from the hilus reaching to the diaphragm; particularly on the right side, but also on the left there were many strands of fibrous tissue although the diaphragm in the fluoroscope showed not a marked grade of limitation. The lungs did not show the type of consolidation which usually accompanies a tubercular parenchymal type. It was somewhat speculative as to whether the bronchial glands were of tubercular origin or not, and the Röntgenologist suggested if the sputum showed any acid fast bacilli, he would probably believe these glands might be tubercular, and the sputum would come from a breaking down of these glands rather than a true pulmonary solidation.

Sputum examinations were made at different times of this individual, which on all occasions showed negative to the finding of tubercular organisms.

The blood was negative on admission as to the number of red cells, white cells, differential count and hæmoglobin. About two weeks after his admission to the hospital his temperature became somewhat irregular, ranging in the morning from a normal up to 102.5 and 103 in the afternoon at four o'clock. At this time a blood count was made and showed that the white cells were increased up to 10,200 his red blood-cells being present to the extent of 4,100,000; hæmoglobin was normal. The urine showed a few granular casts and a trace of albumin. Another sputum examination was made which showed no tubercular organisms but many streptococci and staphylococci and a moderate amount of pus cells. The test for tubercular germ was made by the ordinary method and also by the antiformin method.

Not having found a satisfactory reason for the temperature, an

investigation of the previously complained of right sided pain was made by doing the Lyons-Melzer duodenal bucket test of the gall-bladder. This was found negative from the standpoint of finding any organisms in the gall-bladder.

The possibility of a kidney circumstance in the right side was considered and because of it a cystoscopic examination was made of the bladder and ureters, keeping in mind colon bacilli infection or possible tuberculous infection. The report showed many red cells from the left ureter, a few white blood-cells, no tuberculous organisms. The same report was obtained from the left ureter. Examination of bladder and kidneys showed no urethral strictures. Urine was highly colored. Prostate was moderately enlarged. At the exit of the bladder there was some increased blood supply in the way of a moderate inflammation. Catheter passed up the right kidney readily, urine dropped at normal rate. Pelvis not enlarged. In the left, the catheter passed with some difficulty but reached the pelvis of the kidney. There is no dilatation of the pelvis. Function of the left kidney is fourteen minutes; function of the right is nine minutes, with phthalein injections intramuscularly. Both function times are within normal limits. There is no evidence of ulceration or evidence of tuberculosis of the bladder.

The patient came from the southern Illinois district, where malaria is to be found, and as a consequence, an examination of the blood was made for the presence or absence of malarial parasites. Two slides were made, but no malarial parasites were found. A blood culture was made after the patient was in the hospital eighteen days and showed no growth after forty-eight hours incubation.

The Wassermann was negative.

Up to the present time there was nothing in the physical examination and laboratory examinations from which we could gain a clue to the reason for this man's failing and for the presence of the persistent temperature. It was suggested that the glands in the supraclavicular region be excised and a pathological examination be made on the basis that there might be proved something of interest and importance in the diagnosis of this case. This was done under local anaesthesia; five separate pieces of gland tissue were removed which seemed to be fairly well encapsulated. Color was yellowish-white, consistency was soft and fleshy and there were no caseous areas found

in any of the pieces. Pathological report showed localized areas of granulation tissue of varying degrees of maturity, and composing about one-third of the total area of the section. In places, round and oval spaces lined with endothelium were quite numerous. These spaces were sometimes empty, sometimes contained an occasional small round cell and in a very few of them an occasional red blood-cell was seen. In the areas of most mature granulation tissue, localized collections of lymphocytes representing the remains of the lymph-node. About two-thirds of each of the sections was composed of lymph-adenoid tissue which is diffusely infiltrated with cells resembling fibro-blasts and with smaller numbers of very large cells with from one to three large nuclei. There were no areas of necrosis or caseation.

Diagnosis was made of chronic lymph-adenitis of granulomatous type classified as Hodgkin's disease.

Five days after the removal of the glands from the neck, the temperature persisted, ranging between 97.4 and at the highest point, 103 in the afternoon, gradually slipping down to 102, 100.4, 99.6 on the sixth day. The high afternoon temperature ranged about 99 and about the eleventh day the temperature came to normal and remained there for the next four days, when the patient was discharged.

---

### ACUTE HODGKIN'S DISEASE FOLLOWING HERNIOTOMY

J. P., age thirty-five, male, Austrian, miner.

*History of Present Illness.*—June 26, 1920, was using a crowbar in a mine, when slate fell from the roof. In dodging the slate he fell on a pile of rock. Patient suffered severe pain in right inguinal region, abdomen and chest, and he vomited.

Company surgeon who examined him at the time of the accident noted that the chest and abdomen were negative, but found a right inguinal hernia, extending through the external ring.

Operation June 28, 1920, ordinary right inguinal herniotomy. Before operation usual heart, lung and abdomen examination—with no signs of anything contra-indicating operation. Urine was negative for albumin and sugar. He said he had some pain in his chest, but a careful examination showed no evidence of injury.

Discharged from the hospital in two weeks, operative scar healed and in good general condition.

Two weeks from date of his discharge he again reported to the hospital complaining of great pain in the region of the spleen.

*Physical Examination.*—Showed in areas over the lower posterior left lung—dullness on percussion, bronchial breathing, increased voice sounds and many moist râles. Sputum: Blood tinged; tuberculosis, negative.

*Spleen* greatly enlarged, palpable but extremely painful.

*Blood*, August 4, showed reds, 4,790,000; leucocytes, 15,000; hæmoglobin, 70 per cent.; differential showed small mononuclears, 63 per cent.; large mononuclears, 9 per cent.; polymorphonuclears, 24 per cent.; basophiles, 1 per cent.; unclassified, 3 per cent. On August 14, reds, 3,400,000; whites, 15,600; hæmoglobin 50 per cent. August 18, reds, 2,750,000; whites, 15,600; hæmoglobin, 45 per cent.; differential small mononuclears, 61 per cent.; large mononuclears, 13 per cent.; polymorphonuclears, 25 per cent.; eosinophiles, 1 per cent.

The anemia as noted, grew progressively worse. The lymphatic glands in the cervical axillary and inguinal regions enlarged, not adherent, not painful, did not suppurate.

Temperature was, from August 3 to August 26, irregularly between 100 and 102; August 26 to 31, between normal and 100; September 1 to 5 normal; September 6 to 21, between normal and 101.

Dyspnœa developed and was marked. Spleen enlarged and painful. Death September 21, less than three months after hernia operation.

*Post-mortem.*—Showed broncho-pneumonia, left lung; chronic adhesive pleurisy; enlarged spleen weighing 3½ pounds, marked enlargement of the mediastinal and abdominal lymphatic glands.

*Specimen of Glands.*—Gradwohl Laboratories, St. Louis; markedly infiltrated lymph glands with some lymphocytes, fibroblasts, giant cells and eosinophiles, characteristic of Hodgkin's disease.

The diagnosis in this case before death was acute miliary tuberculosis, although the sputum was negative for tuberculosis on many examinations. This case was in a small hospital in the coal mining district of southern Illinois, so all laboratory tests had to be sent to St. Louis.

At the post-mortem a diagnosis of Hodgkin's disease was made as the glands were discrete and not suppurated and there were no tubercles present.

*Comments on These Cases.*—Etiology of Hodgkin's disease is

still not proven. Bunting and Yates have done much work on this, and have shown that a diphtheroid organism is usually present in the glands, but have not proven this as a definite causative agent.

*Onset* is insidious and the patient usually notices first an enlargement at one side of his neck. This is shown in the first case although it is evident from the X-ray that the mediastinal glands were enlarged first.

The disease may begin acutely, and Zeigler reports cases of death within a month. This is the type of the second case.

*Symptoms.*—Enlargement of the superficial lymph glands was present in both, but the mediastinal glands were also enlarged first in both cases.

Spleen enlargement was also present, and extremely painful in the acute form.

Relapsing pyrexia was present in both cases, the temperature ranging up to  $102^{\circ}$  at times, and remaining normal for some days at certain periods.

*Blood.*—Red blood-cells at first are normal, as seen in both cases; later there is an anemia. The first case showed a drop to 4,100,000. The second showed an acute anemia going from 4,790,000 to 2,750,000 in two weeks. It is possible to have an increase in the number of reds, in cases with intrathoracic growths causing cyanosis and dyspnoea.

White blood-cells may first be unchanged; later a moderate increase to between ten and thirty thousand. The high white count in the first only reached 10,000 and in the second 15,000. There is nothing characteristic in the differential counts.

*Diagnosis.*—The disease most often confused with this is tuberculosis, and both of these cases were at one time so diagnosed. In the second case it closely simulated acute miliary tuberculosis and a diagnosis was not made until autopsy.

The diagnosis is made by an excision of the glands under local anaesthesia, and a careful histological examination which shows changes markedly different from tuberculosis.

*Medico-Legal.*—In spite of the fact that trauma has no causal relation to this disease, both of these cases claimed accidents, and drew compensation for a time.

In the second case a claim for a death award was made on the

grounds that the man had the condition at the time of the hernia operation and that the depression and shock of the anæsthetic and operation aggravated the condition, caused it to become acute, running to a fatal termination.

The glands at the post-mortem were excised, put in a state board of health mailing container, not sealed but using a screw-top cover, sent to a St. Louis Laboratory and there examined. It is interesting to note that this evidence was ruled out as it was held that this container could have been opened and that there was no way to prove that the same tissue arrived at St. Louis as was sent.

---

### SARCOMA OF THE FEMUR

R. B., age sixty-four, laborer.

*History of Present Illness.*—One year ago this man slipped on the steps of a baker shop where he was employed and fell down ten steps, striking his right hip and then rolled down the remaining five or six steps. He got up and continued to work that day, came to work the next day and worked until noon. During this time he had considerable pain in his right hip and it grew worse until he had to stop work. He went home to bed and called his family doctor, who examined him and found that the right hip was swollen and the right buttock was black, but that he could walk except that he had a good deal of pain. This pain remained constant down the back of his leg and he was treated at home for rheumatism and sciatica for several months, walking with crutches and a cane at times, but with considerable pain. Five months after the accident, he was taken to the hospital for an X-ray, which showed no fracture; and later his right sciatic nerve was injected with alcohol without much relief from the pain. Another X-ray was made ten months after the injury which did show a fracture of the neck of the femur and a fuzzy periosteal growth involving the neck, head of the femur and the acetabulum. (See Fig. 7.)

Two Wassermann tests were made and found negative.

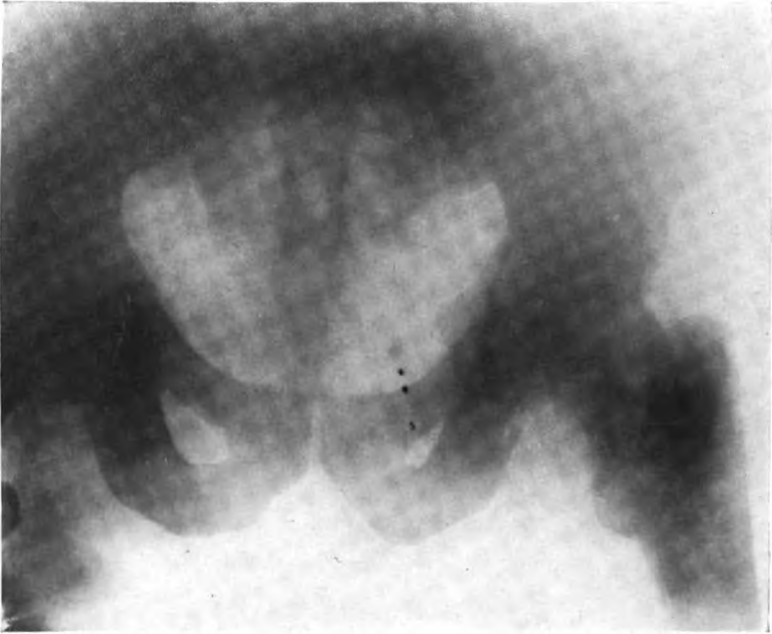
*Past Medical History.*—Patient had jaundice fifteen years ago; no other illness or accidents; denies venereal disease.

*Physical Examination.*—Physical examination shows an old man, lying in bed with his right leg flexed at the hip and the knee adducted and rotated internally. This position he says, is the only one in which he has any comfort.

Pupillary reflexes are normal. Nose and throat are negative. He has false teeth.

Examination of the chest shows the lungs are negative. Heart muscle sound is weak. His arteries are all sclerosed. The abdomen is negative.

FIG. 7.



**Sarcoma of the femur—involving neck and head of the femur and acetabular region showing a pathological fracture of the neck and a fuzzy appearance showing periosteal bone production.**





His right leg from the anterior superior spine to the internal malleolus measures  $34\frac{1}{2}$  inches and the left leg,  $35\frac{1}{2}$  inches. Movement of the right hip joint shows that flexion and extension are possible by passive motion to their normal limits. Internal and external rotation are impossible on account of the severe pain.

This man was requested to come to the clinic for an accurate diagnosis, but he refused to do so and remained at home. The last X-ray was only taken about ten months after the injury. The first X-ray, while it is dim, shows that there was some enlargement around the acetabulum which, at the time, as it was taken some months after the injury, was thought to be callus formation from an injury to the acetabulum. Ten months after the alleged injury the X-ray that was taken shows undoubtedly that the condition is a sarcoma and at that time the patient had a palpable, spindle-shaped tumor involving the upper end of the right femur and the right ilium, and had lost much weight and strength.

He died about a year after the injury, from cardiac failure due to his chronic myocarditis. No post-mortem was permitted.

*Diagnosis.*—In considering the diagnosis of osteo-sarcoma, there are three aids to diagnosis: (1) Clinical symptoms; (2) X-ray picture; (3) exploratory operation with microscopic examination of a section of the tumor.

In considering the clinical symptoms, it should be remembered that pain is the predominant symptom of onset; this pain is usually localized and of a severe, boring character. Localized pain in a bone should be looked upon as an absolute indication for an X-ray examination of the bone. This pain is often the first indication of the presence of an osteo-sarcoma and it is to be noted that in this case pain was the first symptom, and in spite of the fact that pain followed trauma it was five months after the accident that this man was X-rayed. There may be an irregular temperature present but this was not noted in this case. There also may be a leucocytosis with an eosinophilia. Bence-Jones protein reaction in the urine may be present if it involves the bone marrow. Cachexia soon develops and the patient loses rapidly in weight, which this patient did, although no record was kept of his weight.

The second X-ray in this case showed there was a pathological fracture of the neck of the femur. This fracture was not present

in the first X-ray taken. This X-ray is too dim to be reproduced but shows clearly that there was not a fracture of the neck of the femur. Pathological fracture in a bone tumor is usually a symptom of a bone cyst, but in this case the X-ray shows no evidence of cyst formation.

*X-ray Examination.*—From a study of the X-ray in this case the diagnosis was made of a periosteal sarcoma that involved the neck of the femur first, and had extended up into the acetabulum, producing new bone formation in this region. Bloodgood in the "Diagnosis and Treatment of Benign and Malignant Tumors of Bone" in the *Journal of Radiology* for March, 1920, calls attention to the fact that the most common type of periosteal sarcoma shows a typical formation of periosteal bone or a light fuzzy shadow in the X-ray. This is seen in the X-ray of this case. This new bone formation may be confusing if there is a pathological fracture, as the callus may give an appearance of new bone formation, but in the first X-ray taken of this case there was the same fuzzy appearance of new bone formation without the fracture. Carcinoma cannot rise primarily in bone, as there is no epithelial tissue in bone. Therefore, carcinoma can be ruled out in this case as this tumor was primary in this region, as a careful examination of the patient failed to disclose any other tumor formation. In Bloodgood's series, periosteal sarcoma was the next common neoplasm to exostosis and was most fatal, giving less than four per cent. of cures.

*Exploratory Operation.*—This man was seen at his home by one of the authors, five months after his injury and as he was in a small town where there were no laboratory facilities, only poor facilities for X-ray and practically no facilities for an operation, he was requested to come to our clinic for examination, which would have included an exploratory operation, to make a definite diagnosis. This he would not do. It is to be noted that the family doctor cut down on the sciatic nerve and injected it, yet made no attempt to secure a section of what was then a palpable mass in the region of the neck of the femur.

*Medico-Legal.*—This case had a definitely proven accident and a claim was made for the full allowance for death on the grounds that the accident caused the sarcoma. The relation of trauma to sarcoma is in doubt, although many cases give a history of trauma. In these cases of trauma, it is of the greatest importance that X-rays

be taken after any history of trauma, even though there is no fracture and that this X-ray examination be continued at monthly intervals until the disappearance of all symptoms. If the X-ray shows any change of bone production or destruction, there should be an immediate exploratory operation to establish the pathology.

---

## FRACTURE OF THE ACETABULUM WITH INTERNAL DISLOCATION OF THE FEMUR

W. H., age forty-seven, male, crane operator.

*History of Present Condition.*—Injured March 12, 1920, by being thrown from a crane, falling twenty feet, striking the ground. He was taken at once to the hospital, where he was unconscious for four days.

*Previous Medical History.*—Usual childhood diseases; appendectomy, 1910. No other accidents. Denies syphilis; gonorrheal urethritis twice, years ago.

*Family History.*—Negative.

*Physical Examination Recorded at the Hospital.*—Right leg: 1 cm. shorter than left, depression over right trochanter, marked tenderness over right hip joint, right foot rotated outward, large mass six by twelve inches on inner aspect of this thigh, beginning just below Poupart's ligament.

Abrasion under right eye, three small lacerations on right side of nose, lacerations at right lower lip.

Patient semi-conscious for four days; after that all reflexes were normal. Head and neck, negative. Chest, no râles. Heart, negative. Abdomen shows appendectomy scar; no hernia. Lymph glands not enlarged.

X-ray, March 29, showed head of right femur driven through the acetabulum; fracture through spine of pelvis; fracture descending ramus of pubis. (See Fig. 8.)

*Urine.*—Small amount of albumin; sugar is negative. Microscopic examination shows a few red blood-cells and a few hyaline and granular casts, few leucocytes, on March 12.

*Blood.*—March 13, reds, 4,860,000; whites, 9,200; hæmoglobin, 90 per cent.

Discharged from hospital June 4, 1920.

Examination six months after injury at our clinic showed he walked with a marked limp. The right leg was one inch shorter

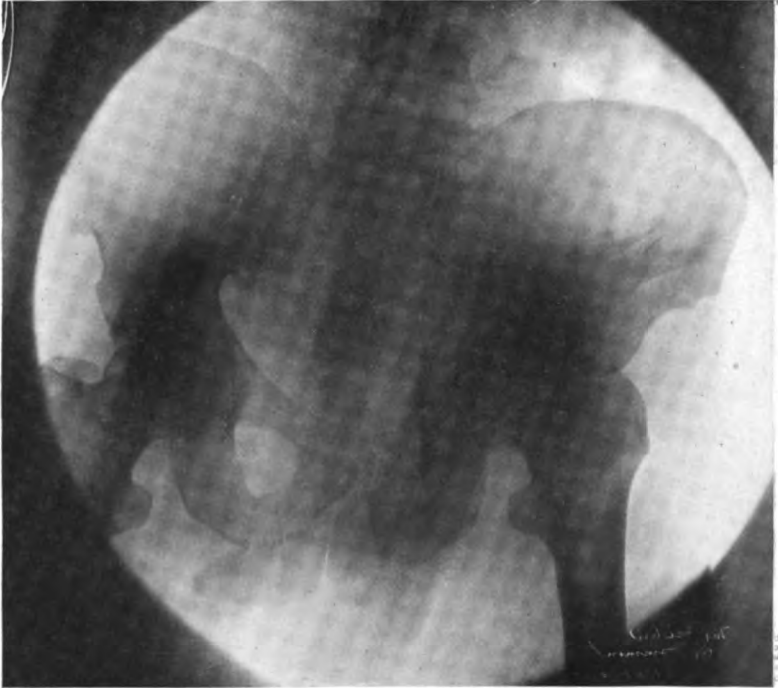
than the left. There was a depression over the great trochanter of the right femur. The motions of the right hip joint showed that abduction was practically absent; adduction was normal but painful. Flexion of the thigh on the abdomen was extremely painful and limited. He complained of pain down the inner side of the thigh. External and internal rotation was extremely painful and limited. This man walked without a cane, but he said that he had constant pain in his right hip which, if he walked over two blocks became almost unbearable. The remainder of his examination was negative for anything that would bear on this injury.

*Comments.*—Schroeder, in the *Northwestern University Medical Bulletin*, 1909, collected and reported fifty cases, including three of his own, and since that time less than twenty-five cases have been reported in the literature. The usual symptoms given are those that were seen in this case. The first thing that was noted in the examination of this man at the hospital was that he had a depression over the great trochanter and it is customary to find the trochanter nearer the symphysis pubis than the one on the uninjured side. There is usually little shortening and in this case, it is to be noted that there was only one cm. shortening recorded in the hospital examination after the man was injured. The acetabular fractures apparently did not unite and this shortening increased to one inch after he was up and around. Hip motions of abduction, flexion and internal rotation are usually decreased and painful. The pain is deep-seated in the hip and the pelvis. On rectal palpation a mass may be felt in this region. There may be pain down the inner side of the thigh from pressure or injury to the obturator nerve. The X-ray of course, in this case, shows the extent of the injury.

The most common dangers are injuries to the obturator nerve, large veins of the pelvis, the bladder or to the rectum. It is to be noted while the fracture in this case was extensive, there were no indications of any of these injuries, although on his examinations in our clinic he complained of continuous pain down the inner side of his right thigh due probably to obturator nerve injury.

*Treatment.*—In treatment of this condition, great care must be taken and no manipulation started until we are sure there are no symptoms of this visceral injury. Feces and urine must be examined and if the urine is not passed in eight hours, the patient should be

**FIG. 8.**



**Fracture of the acetabulum with internal dislocation of the femur.**

2017

catheterized to determine if there is retention or a rupture of the bladder.

The main aim of treatment of this case is to detach the head of the femur from the pelvis. As the head of the femur is pushed through the acetabulum, extension will only serve to lock the head further. Therefore, under an anæsthetic and if possible under a fluoroscope, the thigh and knee should be flexed, and by flexion and adduction with the pelvis held rigid, and a sandbag as a fulcrum on the inner side of the thigh, the head of the femur can be manipulated from the pelvis. Usually only a slight force is necessary as there are multiple fractures in the floor of the acetabulum. If the fluoroscope is not available, rectal palpation can be used to determine if the reduction of the so-called internal dislocation is accomplished, but in no case should pressure by rectum be made upon the head of the femur to reduce this dislocation.

After reduction there should be extension applied with the leg slightly adducted and flexed. Early massage should be started and slight joint motion the second week, but the patient should remain in bed for at least twelve weeks and even after this he should wear a weight-supporting apparatus for a month or so, when he first tries to walk, as the head of the femur may be further pushed through the acetabulum as it was in this case after he had walked on it.

## A SKID FOR THE REDUCTION OF DISLOCATED SEMILUNAR CARPAL BONE

By Geo. G. Davis, M.D.

THE "skid" herein described is not new in principle as applied in fractures of the long bones. It is designed to overcome many of the difficulties experienced in open reduction of the dislocated semilunar bone, especially in cases in which the dislocation has existed over an extended period.

FIG. 11.

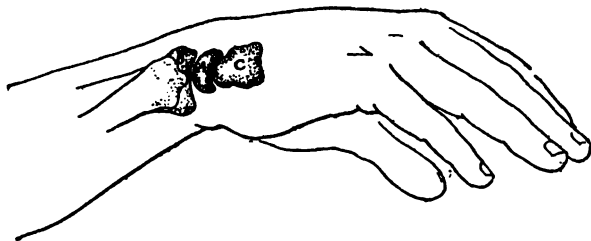


Diagram showing the normal relationship of (a) the semilunar bone to (b) the radius and (c) the os magnum.

FIG. 12.

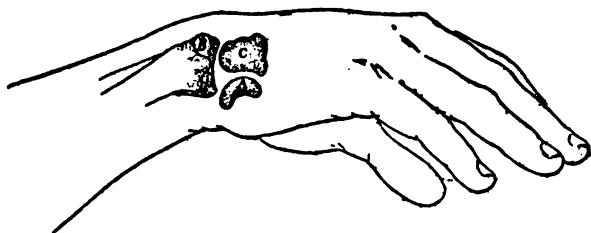


Diagram showing the relationship of (a) the dislocated semilunar bone to (b) the radius and (c) the os magnum.

In the open operation with incision over the dorsal surface of the wrist, the operator finds several obstacles confronting him. There is but little space between the articular surface of the radius and the os magnum, which articulates with the radius when the semilunar is dislocated. With flexion of the wrist and traction on the hand and counter traction of the arm, the space between the two bones is



FIG. 9.



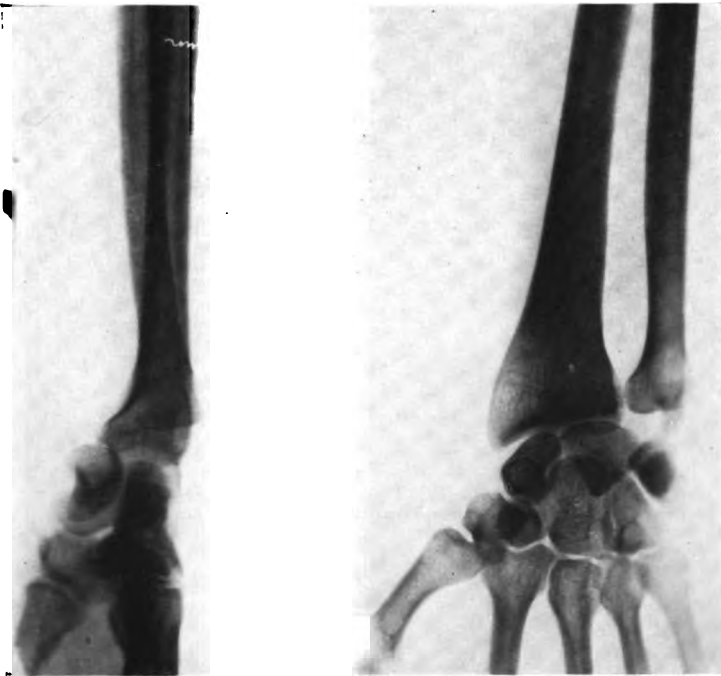
"Skid" for reduction of dislocated semilunar bone—view from above and the side. The instrument is 8 inches in length and tapers from  $\frac{1}{2}$  inch in width at the middle to  $\frac{1}{4}$  inch at the end and varies in thickness from  $\frac{3}{16}$  of an inch at the middle to  $\frac{1}{16}$  at the tip. It has an S-shaped curve at the end. The distal curve,  $\frac{1}{4}$  inch in length, a large arc of a small circle, is to receive the tip of the semilunar bone; the proximal curve,  $\frac{1}{2}$  inch in length, a small arc of a large circle, is to receive the os magnum.

FIG. 10.



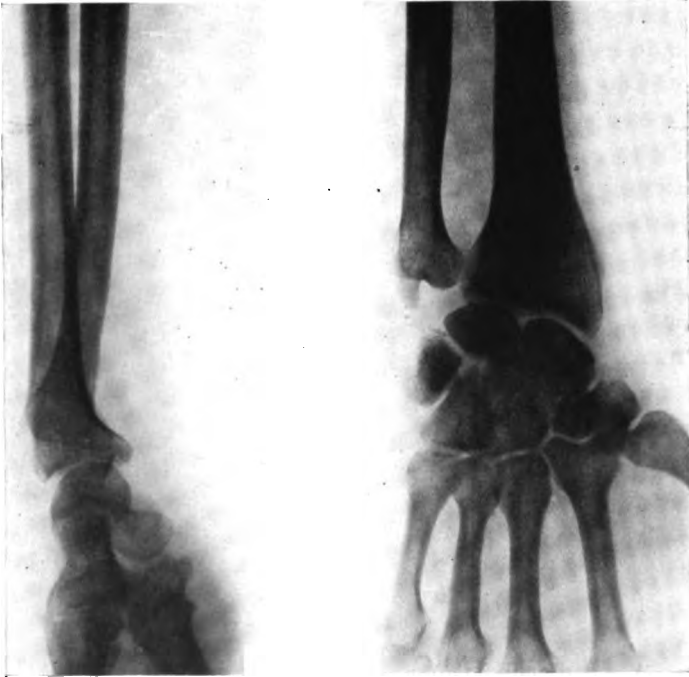
Lateral view of "skid" showing the two parts of the S-shaped curve; the distal curve for the tip of the semilunar bone and the proximal curve for the os magnum.

FIG. 14.



Showing a dislocated semilunar bone in lateral and antero-posterior views. Date of injury September 4, 1920

**FIG. 15.**



Showing the same wrist as in Fig. 6 after reduction by open operation,  
(November 27, 1920) twelve weeks after injury, with good functional result.

increased, but not enough for reduction of the semilunar bone, and the operator now finds himself embarrassed for a suitable instrument to pull or pry the semilunar back into place. There is no instrument in the general surgical armamentarium to meet this special need. A clamping instrument intended to pull the semilunar will damage the bone by crushing. A straight or single-curved instrument applied between the os magnum and the semilunar for the purpose of prying the semilunar into place crushes the os magnum.

FIG. 13.

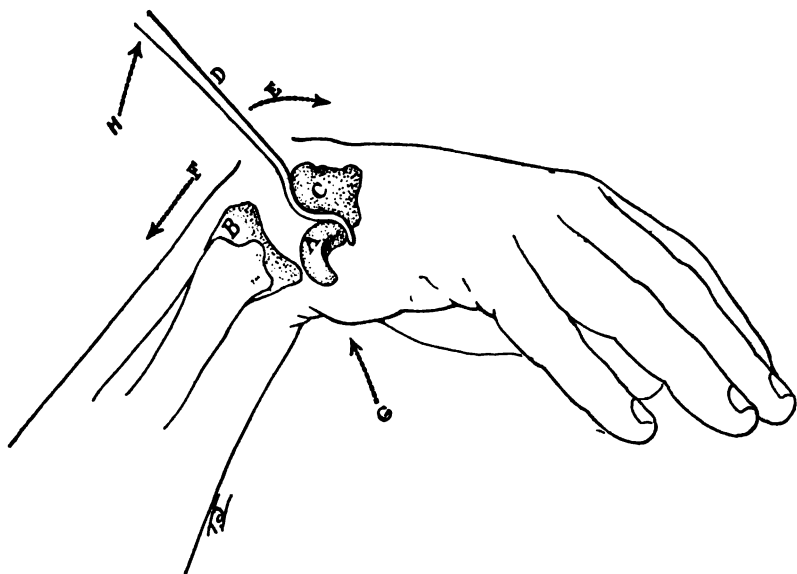


Diagram showing (d) the skid and its relationship to (a) the dislocated semilunar bone, (b) the radius and (c) the os magnum. The arrows mark the direction of the various forces applied to accomplish the reduction of the semilunar. The force applied to the handle of the "skid" is directed distalwards and dorsalwards; the force applied to (c) the os magnum and hand is directed distalwards and towards the volar surface (flexion of the wrist); (f) counter extension is applied to (b) the radius (and arm); (g) pressure applied to the volar surface of the wrist, directed against the semilunar bone.

It is on account of this difficult approach to the semilunar and the resulting injury to the carpal bones that the majority of text-books recommend the removal of the bone rather than the reduction. It is to overcome this difficulty and to save the bone that the semilunar skid is designed.

The skid, which is made of nickel steel, is curved on the flat surface (see Figs. 9 and 10), and is thin enough to slide between the closely

wedged bones. The upper border of the semilunar articulation surface which normally articulates with the os magnum (see Figs. 11 and 12), slides to the volar surface of the os magnum when the semilunar is dislocated. To get the upper border of the articular surface up and back and to apply pressure enough on the os magnum to allow the semilunar to skid back into place, the "semilunar skid" is well adapted (see Fig. 13). The distal curve of the skid engages the tip of the semilunar and the proximal curve skids over the os magnum.

Though the dislocation has lasted a number of weeks, or even months (see Figs. 14 and 15), the reduction with the use of the semilunar skid can be accomplished without injury to the bones.

## ORGANIZATION OF MEDICAL AND SURGICAL SERVICES FOR SMALL INDUSTRIES

By John D. Ellis, M.D.

Associate in Industrial Surgery, Rush Medical College.

---

THERE are, in large industries of Chicago, several very elaborate departments of human maintenance, and the feasibility of establishing such departments in the larger industries is now unquestioned.

Many small industries have some sort of medical supervision. Often this undertakes only the care of injured coming under the Compensation Act. Occasionally, however, as in the particular industries where the statute covering occupational diseases is in operation, some sort of a scheme for the routine examination of employees is being carried out. We will present a brief resumé of the report made on the medical service of an industry averaging about five hundred employees, one of the banks of Chicago, to show something of what can be done in even such a small industry as this, by a rather elaborate system of health maintenance.

This report brings into question the justifiability from an economic standpoint of the establishment of departments for the maintenance of physical efficiency in many of our small industries. The physician is often retained in the first place by the industry with an idea that his work there is to be a quasi-charitable endeavor, and he enters into service of this kind with a considerable handicap because of the viewpoint of employees, in regard to such endeavors directed toward them. We believe that a small industry is not justified in establishing a medical department unless this department is able to give full value received in the maintenance of efficiency and protection of employees from ill health and absenteeism, and through its services actually adds to the work span and the life span of the working force. We question whether industries have a moral or ethical justification in engaging in works of charity toward their employees, and whether the employee is, in the long run, benefited by such endeavor or by such a viewpoint on the part of his employer.

Unless the employee comes to realize that the medical depart-

ment is accessible to him as an aid in the proper care of himself and in the prevention of various causes of disability, the department has failed to realize its greatest possibilities.

Another reason for presenting this short resumé is to point out a scheme that we have profitably employed in developing a medical service for a small group of working people by means of a medical team, in the operation of which one physician calls at the industry each day and examines new employees, attends to certain minor surgical cases, cares for such employees as are ill and decides whether they should be excused from work, and also examines employees who have been sick and are returning to work after the disability; while another man, engaged only in hospital practice in one of the large hospitals where there is present every facility for the care of major surgical cases and for the diagnosis of the more obscure cases, sees such cases as are referred to him by the physician visiting the industry.

#### EXAMINATION OF NEW EMPLOYEES

During the first year of the service of the medical department, from February, 1920, to January, 1921, inclusive, by reason of the shortage of help and difficulty in obtaining properly trained employees for the various departments of the bank, the department heads were especially anxious not to have any applicants for work rejected if they met with the requirements as to special ability or training for the work to which they were assigned. A physical examination of the applicant for work was for that reason not made during the early part of the year mentioned. At the time this report is submitted, however, all applicants are being examined prior to their employment at the bank, as it is now feasible to reject for employment any applicant whose physical condition is found to be such that he would not be able to continue in the service of the bank for any length of time, so that the initial cost of training him for special service would thus be wasted. It is understood, of course, that no employee will be rejected for employment by the medical department, after the routine examination, unless he presents some such physical handicap as will make it impossible for him to become a valuable employee, either because of some progressive disease such as advanced nephritis, diabetes, etc., or some communicable disease

which renders him a source of danger to the health and efficiency of fellow employees such as is instanced by pulmonary tuberculosis or acute venereal infections. The examination of new employees is in reality serviceable to the bank not so much as a method of rejecting applicants for work as by being a method of promoting physical efficiency through the finding and suggesting of proper medical treatment for beginnings of disease and the causes of future disability and absenteeism and the lowering of efficiency, *e. g.*, in cases such as errors in refraction, making it difficult for an employee to use his eyes in close work; or chronic sources of infection and ill health such as may be found in the nasal sinuses, about the teeth, or in infected tonsils, etc.

#### EXAMINATION AFTER ILLNESS

It has been a rule whenever possible to see employees on their return to work after an absence due to sickness, so that the cause of the disability could be determined and suggestions made for the alleviation of the condition or the preventing of future absenteeism from the same cause. The total number of employees reporting to the medical department for examination on return to work during the year was 313.

#### EMPLOYEES REPORTING ILL WHILE AT WORK

All employees who become sick and unable to continue at work have been asked to report to the doctor's office to see whether the condition is something transient which could be relieved by simple emergency treatment or again as in the case of dysmenorrhœa or nausea, by lying in the rest room for half an hour or so with the proper administrations by the nurse. This means of relief of minor ailments has kept a good many employees at work, who otherwise would have gone home for the rest of the day, and who, as often occurs, stay at home an extra day or so before reporting back.

The total number reporting to the doctor's office on account of sickness while at work was 940, of which number 99 were sent home after examination at the doctor's office. Of this number, eight cases of acute appendicitis (four of which were operated as result of the medical department's advice), one strangulated hernia occurring while at work which was handled as an emergency case because of the eminent danger of death, and two cases of pulmonary tubercu-

losis were found. One of these latter was referred to his family physician, who immediately made arrangements for him to have care in a private tuberculosis sanitarium until he recovered; and for the other (after observation of several months, during which time he lived out of doors and slept in an out-of-door sleeping porch constructed by his parents under the supervision of our nurse) an out-of-door job was secured through the interest of one of the officers of the bank in the case. This boy has gained weight and is now in good physical condition, but we cannot advise that he take up indoor work again. Two well-advanced cases of Bright's disease were found in old employees and both are now living on a dietary regime calculated to conserve their kidney function as long as possible. Two instances of severe types of heart affection have been found engaging in work unsuitable for them and their employment has been changed after consultation with their managers. Four more cardiac cases have been found which simply needed some corrective exercises and careful attention to their habits of living, and are now under observation by the medical department. Such examples are stated merely to illustrate the type of service made possible by the examination of employees while at work or reporting to the Medical Department as sick and unable to work.

#### EPIDEMIOLOGY

An informal survey has been made of the epidemiology incident to the working forces of the bank. Infections of the upper respiratory tract rank foremost in the causes of disability and repeated absenteeism, and predominant in this group are recurrent cases of tonsillitis. It is a specific function of the medical department to impress constantly upon persons suffering from frequent attacks of tonsillitis the advisability of tonsillectomy, both as a means of cutting down their absenteeism from this cause and of preventing the serious ill effects which may arise from repeated attacks of tonsillitis, as well as their responsibility for transmission of infection to other employees by coughing and sneezing during times of acute attacks. Eight employees have had tonsillectomies done at our advice and every one of these has gained in weight and improved in general health. One boy gained fifteen pounds in two months after the tonsillectomy. We have a list of twenty-five more employees whose



health records and throat examinations point to the advisability of tonsillectomy and they will be repeatedly urged to have the condition corrected.

Several employees have had nasal operations at our advice for similar reasons. One case of diphtheria was found working and exposing other employees about him to the disease. Positive diagnosis was made from a culture sent to the Health Department. He was taken home by our nurse and promptly treated by his own doctor who found that two children in the family also had positive throat cultures. Every one in his department and with whom he had associated in the bank for a few days before the diagnosis was made, had a throat culture made by the medical department. No more cases were found.

#### NURSE'S HOME CALLS

One full time nurse is employed at the bank; a registered nurse, a graduate of one of the large hospital training schools. She made, during the year, 375 home calls, the highest number during one month being 55, in March. The purpose of making such calls is to ascertain: First, that the employee is really at home on account of sickness, as we believe that such inspection, as soon as employees understand that sickness is investigated, cuts down the amount of absenteeism reported as due to sickness where it is a question whether the employee is actually disabled or is at home for some other reason.

Second, to find out whether the employee is getting proper medical care, as we sometimes find that an employee has gone home sick and for financial or other reasons has had no medical care. He is then advised by the nurse to have medical treatment if it is necessary, or if he needs simply some nursing care, such services are given by our nurse.

Third, calls are made by the nurse for the purpose of attempting to estimate the period of disability, as the employee's manager sometimes feels it is imperative to know whether the employee will be disabled only for a day or so, or for a longer period, under which circumstance it is necessary for him to replace the disabled person or train someone else to assume his functions in the bank.

Fourth, visits by the nurse often help to stimulate the loyalty of the employees to the bank when they realize the institution is inter-

ested in their condition and wants to know they are properly cared for during illness.

#### MINOR INJURIES

A routine part of the service has been that of dressing many minor injuries and surgical affections occurring either at home or while at work, and ranging in importance from lacerations of the hands and feet to such minute injuries as pin pricks, any of which however, even the most trivial, is capable of, and without surgical care often does develop into an infected wound, serious enough to cause disability. Many furuncles and a few carbuncles have been incised under a local anæsthetic and dressed daily until healed. Hardly a day passes that one or more employees do not report for the removal of foreign bodies from the eye, some of which are so deeply embedded in the cornea that the eye must be anæsthetized before they can be extracted. All of such cases may result in absenteeism if not properly cared for. Particularly notable from the standpoint of preventing disability have been many cases of infections and minor injuries of the feet and also cases of beginning flat foot, which have received proper medical attention without the necessity of laying off from work to secure expert professional care.

Our nurse has arranged for application, where necessary, of hot boric dressings several times during the working day, to abort beginning infections. The total number of surgical dressings applied by the medical department during the year was 656. There were 305 cases of infections and slight injuries, not in any way due to work, cared for in the medical department. Seventy-three minor injuries occurred to employees while at work and investigation of these accidents was made to prevent a recurrence of similar cases. This investigation resulted in the following suggestions:

(1) Because of the frequency with which employees have sustained minor burns of the hands while turning on the current to the lights and also to the electrically operated computing machines, that no one be allowed to open or close any light switch in the switch boxes or change the current operating the machine from one plug to another except employees especially instructed in the operation of these.

(2) That the switch boxes controlling the lighting of the bank

be provided with automatic safety switches making it impossible to short the current through the hand of the man operating the switches.

(3) Because of several minor accidents, that the younger boys be cautioned against scuffling or wrestling or flipping pins or paper clips with rubbers. One boy sustained a rather serious eye injury during the latter amusement.

(4) Because several have been injured by upsetting file cases, it is recommended that it be the special duty of some employees to inspect the cases periodically and see that they are securely placed.

#### CASES FOR THE CONSULTANT

During the year nine cases were referred to our consultant for study and care of a type not available at the office in the bank. Among the cases of special interest in this group was a duodenal ulcer, three appendicitis cases, one complicated by cardio-spasm and fainting spells, and two cases of thyreotoxicosis.

During his daily calls at the bank the doctor made a total of 786 physical examinations.

The total number of cases including those dressed for minor injuries, seen by the doctor, assisted by the nurse, during the year was 8663.

#### VACCINATION

In compliance with a request from the City Health Department that the employees of the bank be vaccinated to prevent a spread of smallpox, during the threatened epidemic of this disease last spring, 321 were vaccinated in our medical department, and 34 by their family physicians. No attempt was made to vaccinate those successfully vaccinated during the last 5 years. The total number of days of disability resulting therefrom was fifteen and one half. We believe that this is almost a record in keeping down infections resulting from vaccination and this was made possible by the establishment of a routine of hot dressings on all vaccination wounds as soon as the signs of beginning infection presented themselves, by applying these hot dressings in the medical department either continuously or for a half hour or more at a time, several times a day, as the type of infection and response to such treatment indicated. All serious infections were prevented. The employees who remained at home

during the first few days following vaccination were promptly visited by our nurse and encouraged to report back to work and come into the medical department for proper treatment.

#### PREVENTIVE FEEDING

For several years a system of feeding school children who are below par, physically and mentally, and whose weight is below the proper standard for their height and age, has been in vogue in some of the public schools in America and England. We have conducted an experiment in preventive feeding in industry by which we mean giving between meals twice a day, at ten and four o'clock, a drink of malted milk reënforced by certain other concentrated foods of high caloric value, to certain selected employees who are anemic, thin, and in poor physical condition, with the idea of increasing their weight to approximately the weight and height schedule which has been agreed upon by life insurance organizations, as normal for their age. We have had a chance to apply this method to fifteen female employees who were much below normal weight and strength. The average gain in this group was ten pounds. One gained eighteen pounds, and none less than five pounds. We had several other more or less casual cases who did not get to take the malted milk regularly enough to justify any conclusions, several of whom gained considerably in weight and strength. We believe that getting a person back to the normal weight for his height and age, is one of the best ways of insuring against the various infections and types of ill health which result directly or indirectly from the lowering of vitality and general resistance.

#### RECOMMENDATIONS

During the year's service at the bank we made several observations along the line of sanitation and hygiene, and the following suggestions were made:

The ventilation of the bank is far from adequate. We expect a better system in the new building into which the bank will move within the next two years. In the present building, the hot, vitiated, impure air rises to the upper part of the bank, the most poorly ventilated portions being the mezzanine floors. Drafts from electric fans subject many employees, on whom they blow, to recurrent nasal and

respiratory infections. If the windows are opened the same thing occurs. The only practical method of alleviating this condition and improving the areation of the bank which can be suggested without radical changes is that of installing in the upper frames of four to six of the windows on each floor, high powered exhaust fans, which if operated from five to ten minutes during each hour will thoroughly evacuate the foul air and allow purer air from a lower level to take its place.

Ventilation of the toilets is faulty and can only be remedied by an exhaust fan system. One toilet for women on the third floor is too large, and is provided with a couch. This has become a meeting place or rest room for girls who waste considerable time there—certainly an improper type of rest room.

The use of roller towels is prohibited by the State law, and we suggest that they be replaced by towels on a rack fastener, or by paper towels supplemented by hot-air blowing machines which dry the hands very effectively.

We suggest that the cuspidors of the building be scrubbed and cleaned daily with an antiseptic solution.

In winter the humidity of the air in the bank falls far below fifty degrees. This dry, hot air is one of the greatest factors in the causation of many respiratory affections occurring among employees during the months that the heating system is in operation. This could be, in some measure at least, relieved by the installation of a system of humidifiers attached to the radiators and kept filled with water.

The lighting at most of the desks is such that the light strikes the papers in front of the worker at exactly the proper angle of incidence to reflect directly into his eyes. This is not remediable without a complete change in the lighting system.

More accurate work and a greater volume of work with less fatigue, "nervous break-downs," requests for changes of jobs and general dissatisfaction among workers employed at tedious operations such as running computing machines and certain desk work has been obtained in some establishments by frequent short breaks in the work during which the employee walks about, exercises or does some other type of work. We intend to study with the co-operation of certain department managers, the application of these new methods

in some types of employment in the bank, to see whether some of our problems are amenable to this solution.

We are particularly anxious to complete, during the next year, the examination of the entire working force of the bank. We expect in continuance of our plan to call a few from each department in each day for examination until the entire working force is covered.

These details of our activities in one small industry during a year of operation there are quoted to show how a medical department can be organized in even so small an industry as the one in question and can function in most of the important branches of service covered by the medical work in larger industrial organizations.

In arranging a department of human maintenance for a small industry, which for the purpose of discussion we will take to mean one having less than one thousand employees, the plan of work and its scope depends, as in any industrial medical or surgical problem, on the nature and location of the particular industry.

#### THE "PRESENTING SYMPTOM"

The reason that a small industry "calls a doctor" or seeks medical advice as to its needs is generally a particular one. In those states in which employees' compensation laws are in effect the industry or the insurance company to which it delegates health hazard due to industrial accidents, must in the nature of these laws have some arrangements for procuring surgical aid for such cases as come under the provisions of the statute. The physician doing this work for a plant is furnished an entree into the industry and an insight into its health problems. If he is contented merely to dress such accident cases as are sent to him without attempting to interest the industry in a more extensive plan for the supervision of the health of its workers, he has missed a great opportunity to establish a service of value both to the employer and to the employees; and to accomplish a valuable piece of work from the standpoint of the health of the community.

Industries often seek the advice of a physician because of some such "presenting symptoms" as unusual and apparently uncalled for absenteeism among employees, or complaints from employees regarding real or supposed health hazards coming in the category of occupational diseases or industrial poisoning.

## THE HEALTH SURVEY

A physician consulted on account of some such difficulties must in the first place make a survey to ascertain the type of health services needed by this particular industry. This comprises many things; a study of the "turnover of labor" (the status of the hiring and firing situation), of whether a job is selected for the applicant for work based on his ability and physical qualifications, *i. e.*, whether the job is selected for the man or the man chosen for the job; a study of the health records and average of disability from work in the various departments of the industry; the question of the organization of accident prevention (for in smaller industries the employment of a safety engineer is often hardly justifiable from an economic standpoint and this work falls upon the doctor); first aid methods in vogue at the plant; the question as to whether employees secure proper medical aid at home; what type of nursing service is desirable; sanitation at the plant and its environment, ventilation, heating, light and allied problems; disposal of contagious cases and their management in the surrounding community.

These are only a few of the problems included in the working out of a medical service for an industry.

## PRESCRIBING FOR AN INDUSTRY

The physician must next prescribe such a service as can best be arranged to supervise the health conditions and needs that he finds in his survey. The following is given as an example of the plan of a type of health service we submitted in a proposal to a publishing house in Chicago:

A Medical and Surgical Service for your organization would have in view the following purposes:

- 1 (a) To prevent the spread of contagious diseases;
- (b) To relieve minor illnesses at once, thus preventing many employees from going home for a part of the day;
- (c) To discover diseased conditions early and advise as to the proper treatment, thus preventing prolonged absenteeism from neglect of the case;
- (d) To prevent the employment of applicants who have diseased conditions which might endanger their fellow

employees (as tuberculosis), or which would tend to make them ineffective;

- (e) To improve the sanitary conditions of the working place;
- (f) To prevent occupational diseases and accidents;
- (g) To improve the efficiency of the working force by—

I. Showing a human interest in your sick employees through visiting nurse and your doctor;

II. Relieving many diseased conditions which do not cause absence from work but which lower the efficiency of the individual, as bad teeth, defective vision, diseased tonsils, constipation, etc.;

III. Health talks, advice as to exercise and modes of living, supervision of the treatment of sick employees, etc.;

- (h) To reduce malingering—staying away from work and giving a pretended sickness as the cause.

2 The above purposes are accomplished by:

- (a) Medical examination of all applicants for work;
- (b) Medical examination of all employees returning to work after sickness;
- (c) Inspection by the nurses of all employees taken sick while at work and when indicated calling the doctor for these;
- (d) Gradual examination of all the working force to discover possible causes for sickness or conditions of ill health which may become worse because of neglect;
- (e) Referring all obscure cases to the consultant for examination and diagnosis;
- (f) Periodical sanitary inspections;
- (g) Health advice to individuals and to the entire group;
- (h) Visiting certain sick employees by the nurse for the purpose of visiting nurse aid, ascertaining home conditions, reporting to the doctor as to treatment of the case, its progress, etc.;
- (i) Supervision of the work by your medical consultant;
- (j) The work to be under the direction and supervision of your consultant with an assistant for the routine examinations and visits.

3 This medical system will require:



- (a) A two-room office, waiting room and examining room, also a rest room provided with cots;
- (b) A well-trained, full-time, industrial nurse;
- (c) A trained physician in charge who reports at the industry one hour a day, preferably 9 A.M. to 10 A.M., and is on call at other times of the day;
- (d) Consultations, inspections and supervision of the work by your consultant, who is also always on call;
- (e) Office equipment, card index, files, etc.

## THE DETECTION OF STEEL IN THE EYE BALL

By Charles C. Clement, M.D.

---

THE bad prognosis usually given in the case of an eye that has suffered a penetrating wound and is known to harbor a piece of steel is frequently necessitated as much by the late date at which the correct diagnosis is made, as by the nature of the injury. It thus becomes apparent that an early diagnosis is of much importance, if a larger percentage of eyes having sustained such injuries are to be saved.

The history of the accident and the character of a man's work will often give a clew as to the nature of the injury. The use of power-driven machinery from which small pieces of steel are frequently thrown off with great force, produces many of these injuries, though perhaps a majority of them result from blows struck by a hammer, either in the hands of the injured man, or someone in his immediate vicinity. Not a few result from the clumsy attempts to make household repairs with tools with which the operator is unfamiliar. The variation of steel injuries, each with its own particular symptoms are of course innumerable, and it is impossible to know and recognize all of them, but there are certain well-known facts, the remembrance of which will aid in making a correct diagnosis.

It should be remembered that a small piece of steel may penetrate the eye and cause only very slight discomfort—not nearly as much distress as a small foreign body embedded in the cornea—so the absence of pain does not necessarily indicate that the injury is a trifling one. Men have been known to finish their days work after receiving penetrating wounds of the eye that eventually resulted in the loss of useful vision, or of the eye itself.

Case W. S., age twenty-seven, automobile mechanic.

*History of Accident.*—About 11 A.M. the 1st of August, 1919, while watching a fellow workman hammer a steel nut, a spark flew up and struck him in the left eye. As it caused only slight discomfort, he did not quit work and did not report the accident to his employers. The eye never became inflamed and he lost no time from his work.

In February, 1920 (six months later), he applied for a position with a large electrical equipment company which takes the vision of all employees. He then discovered he had very poor vision in his left eye, although he had had normal sight when examined about a year previously.

*Examination.*—February 18, 1920 (about 6 months after accident). Small scar above centre of cornea. Lens, semi-opaque. Black spot, apparently steel, in about centre of lens. Vision, 20/200. No siderosis. Patient was informed of his condition and advised to consult employer as to determining liability.

March 4, 1920. Pericorneal injection, cortex escaping from wound in centre of capsule. Siderosis. Steel could no longer be seen with condensed light or by direct examination with ophthalmoscope, but was discernable by indirect ophthalmoscopy. Radiograph taken for accurate localization.

*Operation.*—Magnet extraction of cataract. Uneventful recovery with normal vision when wearing proper glasses.

Cognizance should also be taken of the unreliability of the power of observation and recollection of events occurring during times of excitement. The fact that the injured man is sure that the eye was struck by a larger object, does not prove the globe was not penetrated by a small particle instead.

Case I. S., age thirty-seven, chemist.

*History of Accident.*—While driving a nail in repairing a stepladder, something struck him in the left eye. He was quite positive it was the nail and that it subsequently fell out.

*Examination* (the following day).—Perforating wound of cornea in lower outer quadrant into which the iris had prolapsed. Traumatic cataract. Vision reduced to shadows. Radiograph revealed piece of steel in the orbit well behind the eyeball, hence no attempt was made to remove it.

*Treatment.*—Prolapsed iris was abscised and a part of broken up lens removed through original wound. Patient made an uneventful recovery.

Though a sliver or piece of steel had unquestionably been removed from the globe, it is possible a second piece (probably broken off the first one) remains in it.

Case J. A. S., age twenty-eight, machinist.

*History of Accident.*—While working at a lathe about three months previously, a foreign body had entered right eye. He was sent at once to a hospital where a sliver of steel was removed from the eye by a competent oculist. He apparently made an uneventful recovery. About one month ago, noticed failing vision in the injured eye.

*Examination.*—Corneal scar at about three o'clock and hole in iris directly beneath it. Incipient traumatic cataract. Siderosis not detected in artificial light, but plainly seen in daylight. Radiograph revealed piece of steel at most dependent part of globe.

*Operation.*—Removal of steel by scleral route. Uneventful recovery. Returned in six months when lens was entirely opaque and was advised to have it removed but refused.

A piece of steel so minute that it is impossible to detect it with the radiograph may have sufficient force to penetrate the tunics of the eye. (I at one time supposed that any piece of metal having sufficient momentum to penetrate the eye must be large enough to be demonstrated by a radiograph, but a case came to observation about two years ago, which changed that opinion.) In this case, the patient had a very small piece of steel in the vitreous plainly observable with the ophthalmoscope. On account of its very small size, a test case was made of it to see if it would be revealed by the X-ray. Numerous exposures were made by a most expert radiologist of broad experience in ophthalmic work and no trace of the steel could be seen on the X-ray plates, though its approximate location was known. That the particle was steel, was apparently proven by the appearance of siderosis a few months after the injury, attempts at the removal of the minute particle having been unsuccessful.

In a general way any injury to the eye sustained while the patient was exposed to the possibility of being struck by flying steel (especially from hammering) from tools either in his hands, or those of another, should be suspected of having lodged a piece of steel in the globe, unless the nature and extent can be determined and such a possibility pretty definitely excluded.

When a man receives an injury to his eye, he immediately and instinctively covers the wounded organ with his hand—usually quite soiled. This is replaced as soon as possible by a handkerchief probably more soiled, and to these sources of infection is frequently added laving in any water in any kind of a receptacle that is at

hand, so that the eye has had every chance to become infected before it reaches the physician.

Case J. T., age forty-six, stock-keeper in wholesale drygoods house.

*History of Accident.*—While opening a box driving a chisel with a hammer, a foreign body flew in right eye. As it caused no great discomfort, he continued to work the remainder of the day.

*Examination* (one day after the injury).—Perforating wound of cornea of right eye, at nine o'clock. Hole in iris immediately beneath the wound. Pupil contracted and would not respond to cycloplegics. Tension below normal. Vision reduced to ability to see hand movements. Aqueous cloudy. Radiograph revealed steel in vitreous.

*Treatment.*—No attempt made at removal of steel on account of symptoms of infection. Given deep orbital injection of cyanide of mercury 1/3000. Every two hours, day and night, given irrigation 1/5000 bichloride of mercury, instillation 2 per cent. mercurochrome, boric acid compress. Every 4 hours instillation 2 per cent. atropine and 2 per cent. ethylhydro-cuprein.

In spite of the most vigorous treatment panophthalmitis developed and the eye was enucleated, without any attempt having been made to remove the piece of steel.

To the above possibilities of infection should not be added others in the course of the examination. The examiner's hands should be sterilized and the neighborhood of the eye cleansed, care being taken not to wash infection into the eye from the face and eye lids. If the eye is known to have suffered a penetrating wound, it is well to shave the brow at the time of this first cleansing, as it makes it much easier to secure a clean field.

If the wound is a large one, the second step in the examination should be the radiograph, for although the eye may be known to have been penetrated by a piece of steel and the presence of that foreign body in the globe considered reasonably certain, much valuable information can be obtained as to its size and shape and location by a good radiograph. If the wound is not so large as to make itself plainly apparent, a careful search should be made with the aid of good illumination to determine its extent and location. If it has gone through the sclera, it sometimes occasions surprising little hemorrhage, and is easily overlooked. If through the cornea, it is more

easily detected, especially if beneath the corneal wound is a wound of the iris or lens. The piece of steel is frequently seen embedded in one of these structures, or occasionally it drops down to the most dependent point in the anterior chamber, where it is almost hidden from view by the overhanging sclero-corneal margin. If the corneal wound is still open and the aqueous escaping, the anterior chamber is of course obliterated. If there is no extensive hemorrhage or opacity of the crystalline lens to prevent a view of the fundus, the fragment can frequently be seen with the ophthalmoscope by those accustomed to using it, though it would probably be overlooked by one not entirely familiar with this instrument. However, only a part of the anterior of the eye can be explored in this way and failure to see the steel should not be taken to indicate that it is not in the eye.

Case J. K., age twenty-five, structural steel worker.

*History of Accident.*—While cutting a rivet, a foreign body struck him in the left eye.

*Examination.*—A wound about 3 mm. long was discovered about 6 mm. to the nasal side of the cornea in the median line. Tension sub-normal. Pupillary reaction good. By means of the ophthalmoscope a piece of steel could be seen to the nasal side of the disc. Accurate localization was made by means of the radiograph.

*Operation.*—Scleral incision made at point nearest foreign body and steel extracted by means of magnet.

Uneventful recovery.

If the injury is a recent one and no penetrating wound can be seen valuable information can sometimes be obtained by noting the tension of the eye. Reduction of tension to considerably below that of the sound eye would point to a penetrating wound and escape of some of the fluid content of the eye, though normal tension is frequently observed after these injuries, as the wound may be valve-like and close without sufficient loss to cause an appreciable reduction of tension.

If the injury is an old one, siderosis or discoloration of the iris should be sought for. In making this examination, the patient should be in good daylight and not artificial light, as the brownish or greenish discoloration can be detected in daylight long before it can be seen by ordinary artificial illumination.

Case A. D., age twenty-eight, machinist.

*History of Accident.*—About three months previously while hammering a wrist pin, was struck in the right eye by foreign body. Was at once sent to oculist under whose care and observation he had been until time of the examination.

*Examination.*—No scar could be discovered on any part of the eye. Slight pericorneal injection, pupillary reaction sluggish, vitreous opacities. Vision reduced to 20/40 normal. Greenish discoloration of the iris plainly seen in daylight, but not discernable in artificial light. Radiograph revealed particle of steel at root of ciliary body at the most dependent point of the globe.

*Treatment.*—Patient advised to return to his oculist and radiographic localization sent to him.

An iritis, irido cyclitis, uveitis or cataract, which cannot be satisfactorily accounted for, occurring in the eye of a workman should always arouse a suspicion of a foreign body in the eye and the more persistent this condition, the stronger that suspicion would become. The fact that the workman perhaps honestly attributes his condition to a more recent and trifling injury should not throw the examiner off his guard.

If the ordinary methods of examination do not lead to a definite diagnosis, the magnet is sometimes employed. The magnet is an aid of great value in the extraction of these fragments of steel, but should not be used for diagnostic purposes except as a last resort and its use is absolutely contra-indicated where the X-ray is available, as it is accompanied by grave danger of doing irreparable damage to the eye and it is frequently a total failure when judged from the standpoint of furnishing reliable information. If used for this purpose, it should be handled with the greatest care. A magnet whose strength at the tip varies inversely with the estimated size of the piece of steel should be used. That is, for a large piece of steel, one would use a small magnet and for a small piece of steel the giant magnet, and the strengths of these may be regulated by the use of long and short tips. It is impossible to give the complete technique of this test in an article of this length, but in a general way be said if the foreign body is supposed to be in the lens, or in front of it, or if a large fragment has passed through the lens, the tip of the magnet is placed on the centre of the cornea and the eye closely watched for any signs of bulging of the iris or lens, as the current is turned on

and off, and the patient is questioned as to any sensations he may experience at these times. If the steel is supposed to be behind the lens (unless it is a large piece that has passed through the lens) the tip of the magnet is placed on a point on the sclera well back of the ciliary body and as near as possible to the supposed location of the steel and the current turned on and off and the patient questioned as before as to sensations of pain. This test is obviously of value only when the foreign body is of such a character that it responds to magnetic attraction. This can sometimes be determined by an examination of the material and tools used at the time of the accident.

The use of magnetic needles and other delicate instruments for the detection of the presence of steel has practically been abandoned since the advent of the X-ray, as the radiograph is much more satisfactory and is always available where the other unusual and little used instruments were to be found.

With proper equipment in competent hands, the radiograph furnishes more valuable information than can be obtained from any other source and all suspected cases should be subjected to this method of examination, whenever it is available. The detection of the presence of small metallic particles in the globe requires good equipment and an operator experienced in this particular kind of work. Unfortunately there are comparatively few operators who have had the necessary training and experience to make their findings very reliable, and a negative report from the average operator should indicate only that he did not find the foreign body and should not be taken as conclusive evidence that it is not there.

Of almost equal importance with determining whether or not the steel is within the globe, is the question of the exact location, as this will, or should, determine the manner in which the case is to be treated. If the foreign body can be seen with the ophthalmoscope, some idea can be obtained of its location, though this method is far from accurate. If the foreign body cannot be seen, there is no way of determining its location, except by the radiograph and in its absence the magnet. The location of the wound, or the apparent direction taken by the foreign body gives little information, as the fragment may be diverted in its course or may change its position. This is especially true if it is in the vitreous, where it usually eventually sinks to the most dependent point. Accurate radiographic



localization is as a rule available only in medical centres, for it requires elaborate and expensive equipment, whose operation requires special training. If such work is not available, one will have to content himself with the best work he can get. Every eye injury cannot be given a skillful radiographic examination and eyes must occasionally be lost on account of this omission, but it seems evident that if more of the suspicious cases were radiographed, there would be not only a great saving in sight for the injured workman, but also in money paid out in compensation by the employer or insurance company.

# **Surgery**

---

## **THE REDUCTION OF FRACTURES OF THE LOWER END OF THE RADIUS**

**By JOHN H. JOPSON, M.D.**

Surgeon to the Presbyterian and Children's Hospitals; Professor of Surgery, Graduate School, University of Pennsylvania, Philadelphia, Pa.

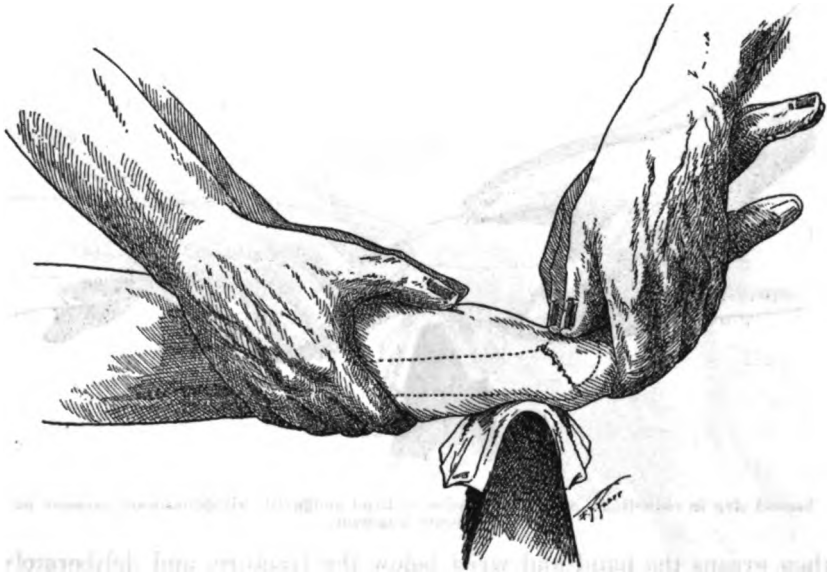
---

**THERE** is apparently no diminution of interest in that common injury, fracture of the lower end of the radius. Papers on the subject continue to appear, largely concerned with the mechanism by which the injury is produced, and the anatomical findings. When the treatment is discussed, stress is laid on the importance of early reduction. The problem of reduction itself, simple as it would appear to be, has not been solved by any standardized method. From our own observations we believe that few fractures go unreduced as often as does Colles' fracture, and that the persistence in many cases of an unsightly and crippling deformity is a reproach to our methods of teaching and practice. It would appear that the methods of reduction as practised are ineffectual in a large percentage of cases of this injury.

For some time we have been in the habit of employing a method of reduction which has been very satisfactory both in recent and old cases, even as late as several weeks after the receipt of injury. While it depends upon no new principles, the details of its application are somewhat different from those in general use. In the first place we would insist on the necessity for the use of a general anæsthetic in all cases, and for this purpose we use either nitrous oxide and oxygen, or the Savariaud mixture (chloroform, ether, ethyl chloride), which was found so useful in cases requiring brief anæsthesia in military practice in France. Thorough deliberate reduction, requiring as it does oftentimes the liberation of impacted fragments, and their replacement in normal relations by manipulation and molding is only possible when the patient is unconscious of suf-

fering, and the mind of the operator is freed from the consciousness that he is inflicting intense pain. For liberation and reduction of fragments the usual principles of primary over-extension of the wrist, to release the lower fragment, followed by forcible consecutive traction, straight extension, and downward flexion, while the forearm is fixed, are the manœuvres required and pursued. For the most efficacious application of these principles it is not sufficient in many instances that the forearm be held and supported by the unaided

FIG. 1.



First step in reduction. Over-extension of the hand and wrist, with forward traction on the lower fragment.

efforts of an assistant. While it is true that in a few cases we can "snap the fragment back into place"—as it is usually described and attempted—the process of thorough reduction is oftentimes much more difficult of accomplishment. We need only mention the suggestion of a very experienced surgeon (John B. Roberts), that a padded hammer be used to pound it back if necessary, to emphasize this difficulty. What is needed is the fixation of the forearm on some form of fulcrum to permit the full deliberate application and combination of extension with leverage, which the hands of the operator

course, by massage, douches, etc. It is necessary to bear in mind the different types of fracture in determining the length of the period of immobilization, and the strength of the tendency toward recurrence of deformity. In fractures involving the articular surfaces, the earlier removal of the splint, and the carrying out of the principle of active mobilization as opposed to the old painful and traumatizing passive movements, must be remembered. The illustrations will depict the method as we have described it. The X-rays illustrate the condition before and after reduction in a typical case. (Figs. 4 and 5.)

**FIG. 4.**



**Typical Colles' fracture before reduction.**

**FIG. 5.**



**Same fracture after reduction.**



## THE RADICAL CURE OF HYDROCEPHALUS \*

By CECIL EDWARD REYNOLDS, M.R.C.S.

Los Angeles, Cal.

IN discussing the subject of hydrocephalus we may, in the language of the zoologist, recognize two species and three varieties.

The two species are:

(1) Those with ballooning of the head—the *obvious*.

(2) Those without visible change of the cranium—the *occult*.

The three varieties are: The Internal, the External, and the Combined. Of the two species, those without visible change of the cranium are overwhelmingly the more important, firstly because they are so often undiagnosed, and secondly, because I am convinced that they constitute a considerable percentage of all epileptics.

I have performed craniectomy upon twenty-three selected cases of epilepsy, which had been diagnosed at one time or another as "idiopathic," and have found decided and often great hydrocephalus occultus, single or combined, in every case.

In regard to etiology, we will, on this occasion, give the spirochæte a rest because, both in hospital and private practice, every case of epilepsy is regarded with suspicion, and if the Wassermann is positive he is helped, if it is negative he can usually go to purgatory on narcotics. I say this in all seriousness because I believe that such is the worst possible routine treatment for epilepsies of organic origin, which constitute probably 60 per cent. of all cases, for reasons which I have published elsewhere. (*Jour. Amer. Med. Asso.*, Sept. 18, 1920, pp. 830, 831.)

The etiology that has come most under my notice has been: Tonsillitis, diphtheria and scarlet fever, typhoid, whooping cough, and a few cases in which a meningitis of subacute convulsive character co-existed with an active gonorrhœa and with no other attributable cause. I feel certain that the germ of poliomyelitis is also guilty, but cannot demonstrate it. The tonsil seems to be the chief

---

\* Paper read and successful cases shown before the Los Angeles County Medical Association, on November 18, 1920.

offender—case after case comes along with the same story of fits after severe tonsillitis or immediately after tonsillectomy, and it is well, therefore, to remember that the ascending pharyngeal artery not only supplies the tonsil but also the basal meninges, and it is the base that is usually the original source of the intracranial mischief no matter how Jacksonian the fits may be.

It is an interesting point that the fits in occult hydrocephalus are very often Jacksonian in character, and almost invariably the left cortex is more affected in the cerebral fits of a right-handed person and vice versa, presumably because the more educated centre is the more sensitive to arteriospasm. In my earlier struggles with this disease I turned down several osteoplastic flaps over the Rolandic areas on account of these misleading focal signs and repeatedly found perfectly healthy membranes waterlogged with clear or yellowish fluid, the entire pathology existing around the fourth ventricle. Conversely, later since I have as a routine measure performed a small preliminary subtemporal decompression in order to equalize the fluid and atmospheric pressures before attacking the base, I have occasionally found gross arachnoiditis with cyst formation and been compelled to fashion a flap with the hinge above in order to deal with the condition. These latter cases, so well illustrated by F. Krause are very much less frequent than general occult hydrocephalus, which has received far less attention.

In the hydrocephalic cases the subtemporal decompression and also the osteoplastic flap occasionally succeed in curing the disease if the dura mater is properly converted into tissue drains, but as a rule no matter how many palliative operations are done above the tentorium, or how efficient the dural or arachnoid drains, I generally have to get to the base before the tonic element of the fits is eliminated, for the tonic element often persists after the clonic convulsions have been eradicated by the supra-tentorial efforts.

Before presenting the patient whom I have selected as an illustration of the radical cure of extreme hydrocephalus, I wish to present again one of the children who were before this meeting two years ago. This child, you may remember, had then been relieved by these operations of some paralysis and very violent fits of which she had suffered five to twenty every night for eighteen months prior to operation, due to combined hydrocephalus



FIG. 1.



The patient (Brendon Cramer) shortly after diphtheria in December, 1917. Observe the typical meningitic facies. He is in center of group.

FIG. 2.



As he was when I first saw him on July 21, 1920. Total paralysis of everything below the eyes including deglutition. Urine contains sugar, and (as almost invariably in these cases) acetone and diacetic acid.

FIG. 3.



Close-up of face shows paralysis of tongue and jaw, the dribbling saliva has been wiped off by an anxious parent.

FIG. 4.



Radiogram by Dr. George Malsbary showing the openings through which the intra-muscular tissue drains were fashioned. The foramen magnum is partly opened as in the decompression operation of Cushing.

FIG. 5.



Somnolent paralysis shortly after final operation.

FIG. 6.



The first successful effort at concerted movement of all four limbs on the fifth post-operative day of the second operation (the basal).

FIG. 7.



FIG. 8.



FIG. 9.



**The fatuous semi-paralytic stage.**

Early acts of intelligence about the tenth day. Note the apraxia of the right hand in this left-handed boy. These defects as well as speech improved by leaps after each post-operative fit. He had diminishing fits for about two weeks after the final operation.

Also note the defective vision before the disce cleared up.

FIG. 10.



FIG. 11.



Early efforts at speech. He took so long framing one syllable that I was able to take a time exposure of his lips. Later he stammered in the familiar so-called functional way. His speech is now fluent.

FIG. 12.



The incisions from the left side. He is left-handed.

FIG. 13.



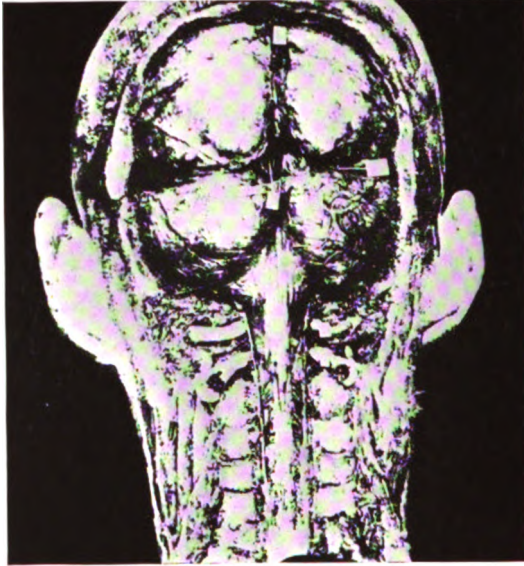
The cross-bow incision.

FIG. 14.



The patient on November 10, 1920. His last fit was on August 5th, mild.  
(This patient recommenced slight infrequent Jacksonian fits in July, 1921.  
Requires a right osteoplastic flap and deduralisation to render cure permanent.  
He is in Chicago now.)

FIG. 15.



The arachnoid covering the fourth ventricle and its pial diaphragm, after Professor Cunningham's dissections. This arachnoid usually remains intact after the dura is reflected from off the cerebellar hemispheres, but when the arachnoid is adherent to the dura reflection of the latter may open the fourth ventricle. In the present case this arachnoid sheath was adherent to the deep aspect of the occipital sinus which I tied and divided. I then intentionally opened this layer of the ventricular roof as I was not convinced that its powers of osmosis had not been impaired, since they were certainly lost from the basal arachnoid that lined the great cistern, which had become like parchment instead of like blotting paper, and I remembered that some authorities assert that there are no such things as the foramina of Majendie, Key and Retzius in the living subject. (For example: Coupin in the C.R. Soc: Biologie, vol 83, No 22. 1920). I do not recommend this as a routine procedure however effective in this instance. Some cases have neither a tela choroidea inferior nor this sheath of arachnoid remaining to cover the fourth ventricle so that when one opens the dura mater anywhere at the base one finds oneself confronted with a great pool or lake which gushes forth with a violence that is only held in check by the iter of Sylvius, and if this is also dilated the resourcefulness of the surgeon is strained. The Rhomboid fossa stares one in the face immediately the dura is reflected covered only by the lobes of the cerebellum.





FIG. 16.

This is such a case photographed in the Clara Barton hospital on April 20, 1920, the day before I did her subtemporal decompression. Her trouble began as usual with severe tonsillitis at the age of seven years in August, 1910. The disease took the usual course—typhoid, then fits and more fits in greater and greater severity and variety until in Feb., 1917, she came under the care of a well-known neurologist in a large Eastern hospital. Again the focal signs, deceived, and the hospital surgeons turned down an osteoplastic flap over the left lower Rolandic area and found nothing but abundant fluid. Unfortunately, the hand of Custum led the surgeon to stitch back the dura mater which is in my opinion, a thing that should scarcely ever be done anywhere at any time. The result in this case was a brief improvement followed by loss of speech, continued convulsions, and increasing paralysis until she became the poor comforted and incontinent dement shown in this picture. After the subtemporal drainage, I turned down the old osteoplastic flap. Beneath the stitched dura was encysted fluid and a yellow excavation in the sensory hand area the size of a pigeon's egg as the result of an old post-operative blood-clot. I wish to lay emphasis on this question of the dura because I have not stitched an intracranial dura for four years and have seen no ill result from allowing a new one to grow, and I also leave the spinal theca open in certain inflammatory conditions. I have seen even worse effects than the above in cases in which the spinal membranes had been closed after injury and after inflammation. One point is essential and that is when the fourth ventricle or the spinal theca is left open, the muscles should be accurately sutured with silk; not catgut.

FIG. 17.



This is the same girl as above, a few weeks after the deduralization. She can sew and do ordinary duties. However, the cerebellar element of her fits persisted and I therefore performed the radical fourth ventricle operation in two stages. She had no walls whatever to the ventricle, but I anticipated this and only made a small hole in the dura at the first stage. In spite of this caution the fluid came out under very great pressure and her blood-pressure went down to 40 systolic. At the second stage the dura was completely reflected displaying a naked Rhomboid fossa which is now covered only by cerebellum and the deep muscles of the neck. The result is that she is now, Nov. 18, 1920, articulating sentences distinctly for the first time for over 3 years and has had no more fits of any kind.



**FIG. 18.**



The same girl a month ago. (Aug., 1921. This girl has developed generalized tuberculosis and is near death.)

**FIG. 19.**



The same girl in 1911 shortly after the onset of her disease. Note the paresis of the lower face which led to the old osteoplastic flap.

FIG. 20.



The other patient presented two years after cure by operation of acquired hydrocephalus of the second degree, that is, the arachnoid of the great cistern was adherent to the dura and had lost its osmotic power, but the roof of the fourth ventricle was apparently in possession of its osmotic power, and was suffering only from back-pressure. She had some paralysis and from five to twenty or more fits every night for 18 months prior to operation. (Aug., 1921. This child still enjoys perfect health and has had no fits since Dec., 1918. She is unusually bright and studious.)

**FIG. 21:**



Photo taken October, 1920, of E. P. whose case was reported in *International Clinics*, vol. III, series 28, 1918. She has been cured of epilepsy since operation in November, 1917.

**FIG. 22.**



**The attitude in paralysis of the anterior vermis. This man had a malignant cyst of the vermis that burst into the third ventricle. Case referred by Dr. F. L. Anton.**

**FIG. 23.**



**Distension of the third ventricle from extreme and neglected occult hydrocephalus, exhibiting the same attitude.**



following tonsillectomy about a week before the first symptoms. I did four supra-tentorial operations and got results from each, but after all cerebral fits, of which there was a variety, had been eliminated, the cerebellar spasms continued until I fashioned intramuscular drains out the dura-arachnoid which, adherent to each other, had covered both cerebellar hemispheres. The coverings of the fourth ventricle were not in this case opened either by nature or at the operation. She has not had a fit of any kind or any ailment whatever since this last operation in December, 1918. She is above the average of her age in her studies, having jumped a grade in school last month, and is very athletic in spite of the fact that both Rolandic areas and both cerebellar hemispheres were partially "denuded" of dura mater two years ago.

The other earlier cases that I have presented are in similar perfect condition.

(The patient, Brendon Cramer, was now presented.)

This boy was born in May, 1915. He was unusually bright and talked at ten months. He and his younger brother both contracted diphtheria in December, 1917. He received ten thousand units of antitoxin, and later became delirious but his brother was unaffected at this time. Ten days later both children had fits of great severity and frequency. Later, when he first attempted to walk he would fall "as if poleaxed" with a sensation of being hit over the right ear, and sometimes with an hallucination of whistling. Then appeared enlarged glands in the neck. The fits became worse, and after being observed in the Michael Reese Hospital, Chicago, he came to California and soon lapsed into a condition of total paralysis, combined with many fits both day and night, in which condition I first saw him in July of this year. As the general paralysis became more profound, the fits lessened somewhat as is the rule when a brain is losing its "kick."

As the physiology of this case fills a volume I will cut technicalities and show pictures of what occurred.

It may be stated, however, that after removal of very bad tonsils in August, 1919, he improved vastly for four months, after which time the trouble recommenced with hiccough and then fits and marked mental disturbance. The fits soon attained a frequency of from ten in the 24 hours to thirty per night of the mixed basal and

cortical type until the paralysis supervened rapidly early in June.

You will particularly observe that he is now mentally bright; in this respect he is making steady progress, and no backwardness is evident without searching examination.

Summary of the degrees of occult hydrocephalus as met with surgically:

1. Excessive fluid in the subdural space both at the base and over the vertex. Arachnoid applied to the pia over the hemispheres but the basal water-bed between arachnoid and pia well filled under the cerebellum. The normal distension of the cisterna magna is hard to standardize.

2. The arachnoid of the cerebellar hemispheres is separated from the pia by abundant fluid and is adherent to the dura, so that the cisterna magna is extended upward over the cerebellar hemispheres posteriorly without becoming continuous with the fourth ventricle, the arachnoid sheath of which appears to have firm lateral attachments. This arachnoid sheath of the fourth ventricle may or may not be pathologically adherent to the deep aspect of the occipital sinus. Arachnoid of the cistern has lost its osmotic faculty.

3. The above mentioned arachnoid sheath of the fourth ventricle has burst its lateral attachments and become continuous with the abnormal upward extension of the great subarachnoid cistern. The tela chorioidea inferior naturally gives way also. The fourth ventricle has now become part of a large subarachnoid lake into which the scalpel will be inserted as soon as it penetrates the arachnoid which is everywhere adherent to the deep aspect of the dura, on attempting to open the latter anywhere over the cerebellum.

4. In addition to the above, the iter of Sylvius is also dilated, and if the condition has existed long enough, the lateral and third ventricles as well. In such cases where the fourth ventricle has no walls, it is advisable to leave the occipital sinus stretching across the cavity like a harp string. It serves as a future point of growth for new dura after the cerebellum may have expanded to its proper dimensions.

The converse cause of distension of the third and lateral ventricles, *viz.*, blocking of the iter of Sylvius, does not come within the scope of the present discussion. I have not met with it surgically in acquired occult hydrocephalus.



## FRACTURES OF THE SKULL—MECHANISM OF THEIR PRODUCTION

By R. J. BEHAN, M.D., F.A.C.S.

Surgeon St. Joseph's Hospital, Pittsburgh, Pa.

IN order that we may formulate some idea as to the frequency and type of fractures of the skull, I shall draw attention to a study by Joseph Wiener (*Amer. Journ. Surg.*, Dec., 1906. "A Plea for Exploratory Incisions in Suspected Depressed Fractures of the Skull"), who studied 62,864 fractures, which he statistically arranged as follows: Eight per cent., or 1139, were fractures of the skull—of these, nine were in men to one in women; 50 per cent. were due to a fall; 35 per cent. were due to a blow; 9 per cent. were due to being run over; 44 per cent. were of the frontal bone; 40 per cent. were of the parietal bone; 14 per cent. were of the temporal bone; 2 per cent. were of the occipital bone; 85 per cent. of these were the result of direct violence and 84 per cent. were of the anterior portion of the skull.

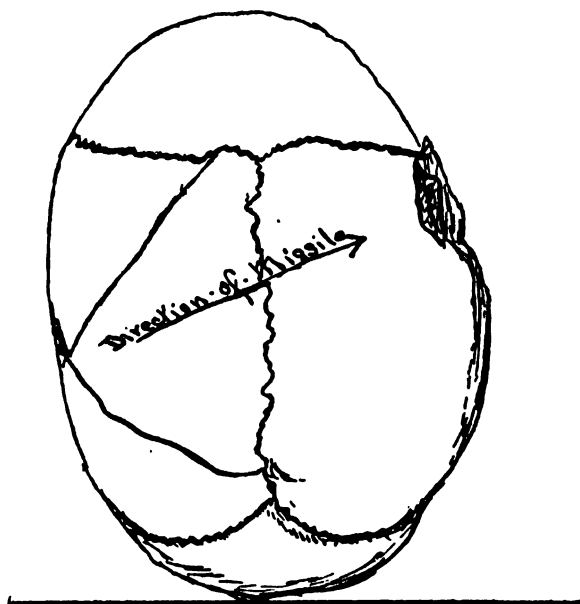
In order to review the mechanics of fracture of the skull, I shall draw attention to the fact that all fractures of the skull are the result of force applied either directly or indirectly to the skull. The direct force may be applied at right angles to the skull or at a tangent to it, or may act as a compressing agent.

The common agencies are: a bullet, a sword, a knife, a pick, black-jack, or a contact of the head with the ground. Depending upon the medium of application, there results a small or a large fissured fracture, a gutter fracture (Fig. 1), a bursting fracture (as shown in Fig. 2), a perforating fracture, or a stellate fracture, and the fracture may or may not be depressed.

As a rule, any force which has been strong enough to ding the skull causes a marked laceration of the soft tissues and destruction of the bones at the point of impact. If it is a bullet or a sharp-pointed object, usually the inner table of the skull is more comminuted and is destroyed to a greater extent than is the outer table. It is claimed that a foreign body (bullet) with the proper momentum and force may pass into the skull and the skull itself be so little

damaged that the opening made by the passage of the bullet can, with difficulty, be seen. Bending-in of the skull sometimes occurs when a blow has been struck by a blunt instrument. The inner table is fractured and comminuted, although the casual examination of the wounded scalp may disclose no apparent fracture. In bullet wounds the fragments of both tables of the skull may be driven in all directions into the brain and the bullet itself may become broken up and

FIG. 3.



This is an illustration taken from the *Manual of Neuro-Surgery* (p. 128) issued by the Surgeon-General. It shows the meridional lines of fracture radiating from the point of impact (entrance of the bullet). It also shows the bursting effect of the bullet at the point of exit.

scattered. On the other hand a spent bullet may ding the two tables and give rise to no fracture. Perforating bullet wounds of the skull generally are the same as perforating bullet wounds in other parts; that is, the wound of exit is larger and lacerated more than the wound of entrance (Fig. 3). Bullets may, however, produce little apparent damage (see below).

In considering bullet wounds, it must not be forgotten that the modern high-powered rifle gives a rotary motion to the discharged bullet. This rotary motion is comparative to the speed of the bullet. However, a spent bullet revolves not only on its longitudinal axis, but

FIG. 1.

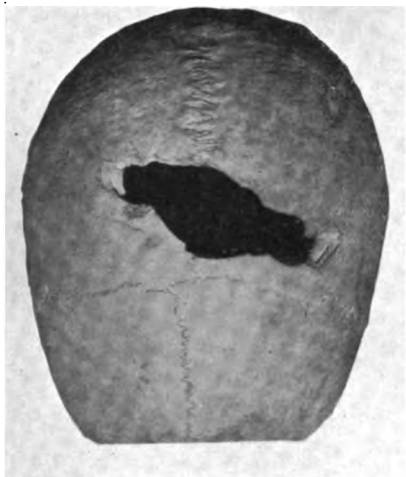


FIG. 2.

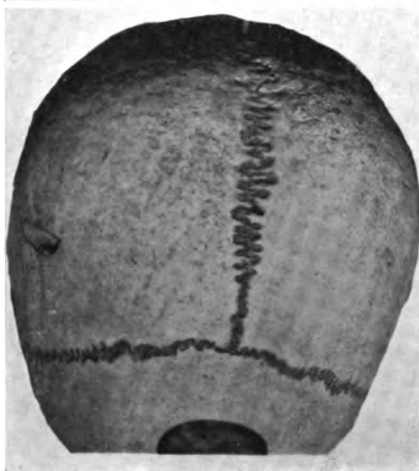
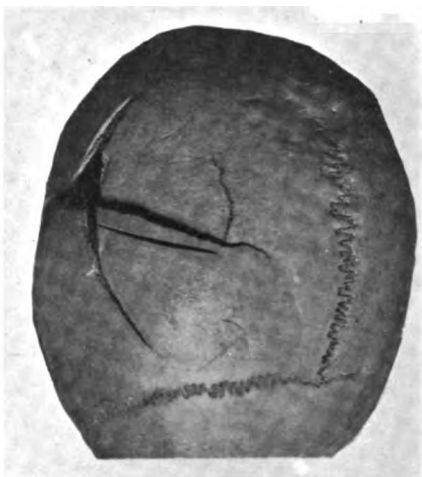


FIG. 7.



FIG. 8.

FIG. 1.—Gutter fracture, due to a small object (bullet) coming into contact with the skull in a tangential direction with great force. (Cornell Collection.)

FIG. 2.—Bursting fracture, due to an object of great extent coming with great force into contact with the skull. (Courtesy of Dr. Ewing, Cornell.)

FIG. 7.—Bullet wound of the skull. This shows the depression of the external table. The next picture shows the fracture and depression of the inner table. (Cornell Collection.)

FIG. 8.—Shows a fracture and depression of the internal table of the skull resulting from the impact of a spent bullet against the external table of the skull. This is a companion picture of FIG. 7, and the appearance on the external surface of the skull of the same injury as illustrated in FIG. 7. (Cornell Collection.)

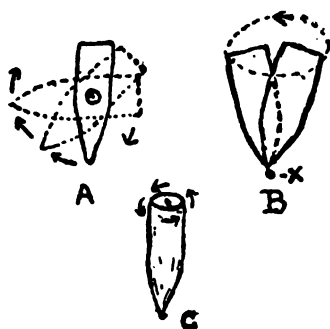


also has a running motion around its apex as a centre, and when the velocity decreases it rotates around a transverse axis, so that an extensive wound results when it comes in contact with the skull (see Fig. 4).

In order to thoroughly understand the principles underlying fractures of the skull, I shall briefly review the mechanics of a simple fracture of the skull:

The skull consists of two tables, with an intervening, spongy, vascular structure called the diploe. This diploe lies between two layers of compact, fairly dense bone, the outer layer being called the

FIG. 4.



The (three) rotating movements of a bullet in flight. A shows how a low velocity bullet turns over and over on its short axis. B shows how a high velocity bullet rotates around a longitudinal axis, the tip of which is fixed at x. This bullet has the motion of a spinning top. C shows how a very high velocity bullet rotates around an axis which is the longitudinal axis of the bullet.

external table and the inner layer the internal table. It should also be recalled that the skull in the greater part of its surface is curved, so that these tables may be compared to two circles superimposed on each other with an intervening space, as shown in Fig. 5.

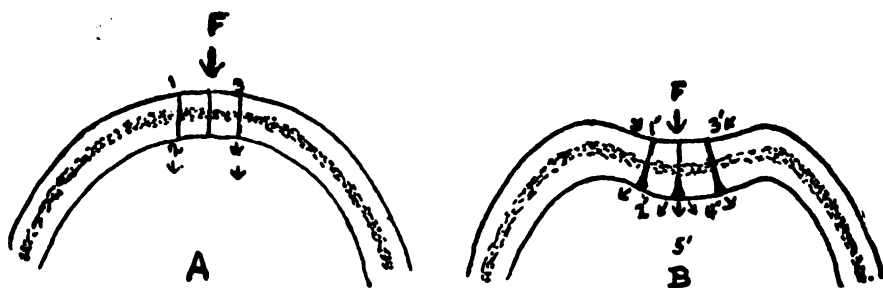
What will happen when a body comes into forcible contact with the skull, depends upon first, the shape of the body, second, the momentum with which it hits the skull, and third, the area of contact.

If the object is very small and comes with great rapidity, as a high velocity bullet, it may immediately perforate both tables of the skull, and pass through the brain and out on the outer side of the skull, producing a minimum of injury to the skull and brain. A nephew of General Putnik, whom I attended during the second Balkan War, had a motor aphasia, due to a bullet, which had completely traversed the skull, without producing much apparent laceration. There was

no perceptible destruction of tissue, except a small wound of entrance and a slight wound of exit. The extent of bone destruction depends upon the shape of the missile, the momentum and direction of contact (see Fig. 6). If the object is small and the force of impact is reduced, as from a spent bullet, if the impinging object is somewhat larger than a bullet and the momentum is decreased, as in a blow from a club, a fracture of both tables may occur at the point of impact. This is explained in Fig. 5.

In *A* the arrow *F* represents the impact from a small object applied with great force. The energy is transmitted almost directly

FIG. 5.



*A* shows a normal skull with the lines of force at *F*. *B* shows the depression resulting from a blow of restricted impact at *F*. There is a gradual tendency for the bone particles (*5'*) opposite the point of impact to be forced apart, so that a fracture occurs at this place. If the force is great enough and the area of contact is sufficiently restricted there also occur fractures at 2 and 4.

through the skull to the underlying brain and thence onward (Fig. 5).

However, if the force is less and time is given for the inertia of the skull to be overcome, the action of the force is to first ding the outer table. As this occurs the molecules in the outer table are forced more closely together. As the force continues the inner table is bent inward. The molecules in this table at the points 2 and 4 tend to become separated, as is noted in *B*. The boundary line 3-4.

A line of fracture will develop in three places (Fig. 5). First where the cohesive power between the molecules is less than the disruptive force. When the cohesion is not sufficient to overcome this disruptive force, a line of fracture occurs at 2 and 4, as in Fig. 5. There is also a great tendency for the cohesive power existing between the molecules also to be broken at a place on the inner table directly opposite the mid-point of the area over which the impact

(force) is received. At this point depression of the inner table reaches its greatest magnitude.

In a fracture of this character, solution of continuity occurs first in the inner table, directly opposite to the area of contact with the impacting object.

If the force is very great, such as that exerted by a bullet, a portion of both tables of the skull may be punched out and driven into the brain substance. Stress also exists at two points—slightly removed from the direct action of the force—and secondary fracture, particularly of the inner table, occurs. (Note 3 and 6 in Fig. 6.) In other instances the force may not be sufficient to cause the object to penetrate

FIG. 6.



The figure shows the result of a bullet perforating the skull. The opening in the external table of the skull is a little larger (in diameter) than is the bullet, but the opening in the internal table much larger. This is the result of the driving through the internal table not of the bullet (or foreign body) but also the fragments of the external table. There is an additional fracture of the inner table at a little distance from the perforation, i. e. at 3 and at 6.

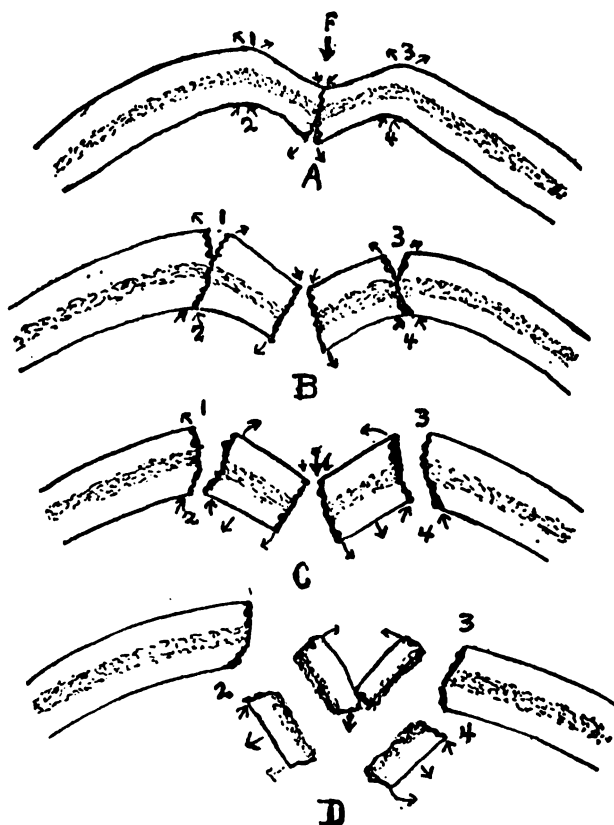
the skull. If the area of contact is small, such as occurs when a bullet or the blade of a pick comes into contact with the skull, both the tables may fracture and ding inward although the agent producing the trauma does not penetrate the skull (Figs. 7 and 8).

Depression of both tables may be just sufficient to cause the molecules of the inner table to loose contact with each other and so produce a fracture of the inner table alone. After the force is relieved this table may spring back into place, so that examination of the external table alone reveals no solution of continuity. This is a most serious type of fracture, as extensive damage may be done to the dura and brain cortex with only slight or no external evidence of such injury.

The progressive phases of a fracture of the skull from a slight bulging to that of marked comminution and disintegration are shown in Fig. 9.

If the object coming into contact with the skull is large and the momentum is sufficient, a depressed fracture of the skull may result,

FIG. 9.



This figure illustrates what happens in the skull when a small object, such as a bullet, traveling at a moderate velocity, comes into contact with the skull. At A both tables are depressed, there is an acute angulation at 1, 2, 3, and 4. Directly opposite the centre of the depression on the inner table of the skull, there is a beginning crack or separation of the internal table. If the force continues we find that the tables are pushed deeper into the cranial cavity, the crack of the internal table widens and there also appears a fissure on the external table at the point of the most acute angulation, at 1 and 3. If this force still continues we find that the fissure has extended entirely through the two tables in the centre and that also there is a separation of the two tables of the central depressed area from the intact portion of the skull, at 1, 2, 3 and 4. At 2 and 4 the tendency is for the fissure to take a direction away from the central depressed portion. Above that, the direction of the fissure is probably in the same direction as when the fissure occurs in the inner table; that is, it is away and directed outward from the inner table to the surface and away from the direction of the depression. Compare above figure with Figs. 12 and 13.

At D it shows how the two fragments come together and the two lower fragments, that is, the fragment of the inner table tend to separate, so that there is an opening formed between the two fragments of the inner table through which the upper fragments pass.

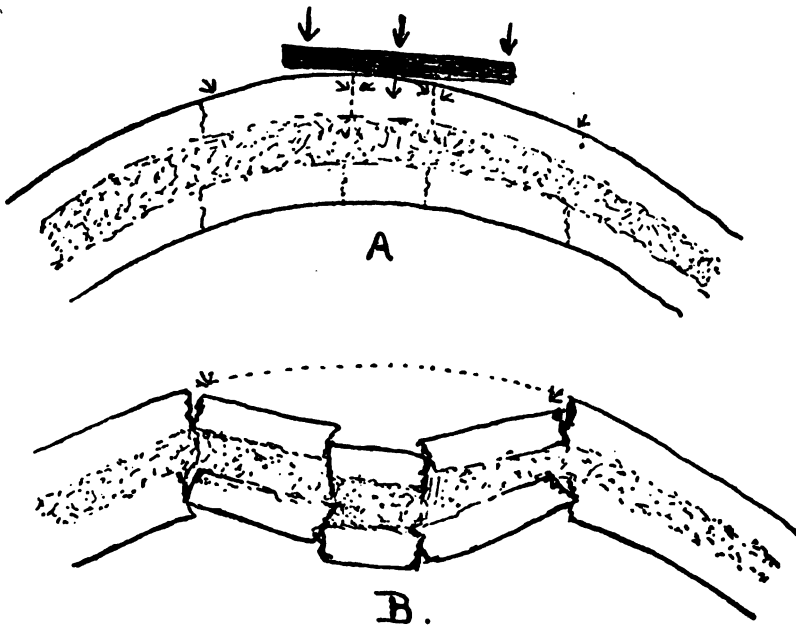
This produces a most disastrous and severe destruction of brain substance. In some instances it acts with the force and destruction of an explosion. If the force (a bullet) comes with sufficient velocity numerous small fragments of bone are driven in all directions so that there is practically a disintegration of the brain.

as in Fig. 10. It is from forces of this type that fractures of the cortex and base of the skull so frequently result.



The severity of the injury to the skull and its contents also depends to a great extent upon the area of contact and the magnitude of the blow. A violent blow coming into contact with the skull over a small area, as a rule, will produce a more severe injury than if the area of contact were greater. A blow of limited contact area and of the same force is more likely to cause an injury both to the inner and the outer table than is a blow of the same degree over a wider area.

FIG. 10.



Shows a depressed fracture, the result of a blow of considerable force over a large surface of skull. The lines of force are as indicated in the figure. The central fragment being first displaced, pushed downward, then the two lateral fragments are broken and forced downward. The figure is diagrammatic but represents the mechanics of this type of fracture.

In the latter class of cases the injury very frequently is confined to the inner table alone. This type of fracture is illustrated especially in the so-called "Ping-Pong" fracture, occurring among children, living in the lower East Side of New York, who, while playing upon the roof, sometimes slip, fall over the side and light on the ground on the head (Fig. 11).

These little patients are brought to the hospital and the operating surgeon finds, as a rule, only a ding in the outer table of the skull. However, on cutting through the external tissues to the bone and tre-

phining the outer table, destruction of the inner table usually is present, though sometimes all that is discovered is a bulging-in of the two tables. In the latter instance, both tables may be sprung into place, by means of a screw or chisel inserted through the bone at the point where the depression is most prominent. Sometimes, if a fissured fracture is present a bone elevator may spring the tables back into place, or if this is not possible a trephine opening may be made at the margin of the depression and an elevator inserted through it, and the bone may be sprung back into place. Such cases may not suffer any bad effects.

A rather sharp instrument, such as a hatchet, sword or an axe may produce dingeing of the skull, with a fracture of both tables (Figs. 12 and 13).

There is also a type of fracture in which extensive fragmentation of all the bones of the skull occurs. This follows the receipt of very powerful blows upon the skull or falls on the head from great distances. In one such case the skull was so fractured that the line of fracture ran entirely around the head, and the upper portion of the cranium was completely forced down over the lower portion, and severe compression of the brain resulted.

#### COMPRESSION FRACTURES

Compression fractures may occur in those whose heads are caught between the bumpers of railway trains or between an automobile and the lamp-post or between a curb and an after-coming sled, etc.

This is illustrated by the fracture resulting from compression of the skull in a young woman, a school teacher, who fell off a rapidly moving sled and whose head was compressed between the sled and the curb. The apparent external injuries consisted of bruises and laceration of the skin over the right orbital and malar bones on the right side and over the prominence of the parietal bone posteriorly on the left. There was no other external evidence of injury. The X-ray showed a transverse fracture extending over the convexity of the skull from the temporal fossa on one side to the temporal fossa on the other side (Fig. 14).

Falls also act as compressing forces, if the person falls upon the convexity of the head. The head is compressed between the floor or the ground and the after-coming body. It is claimed that in such cases the fracture first occurs at the base and then as the compression

Fig. 11.

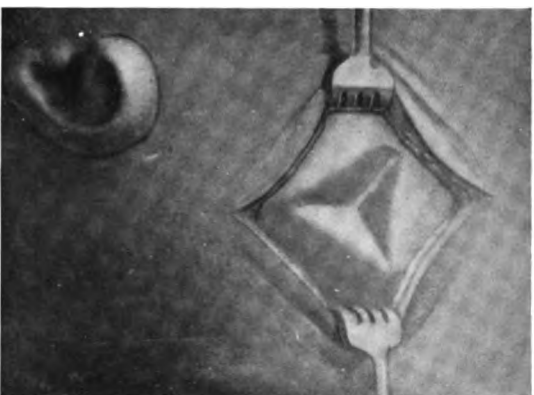


Fig. 12.

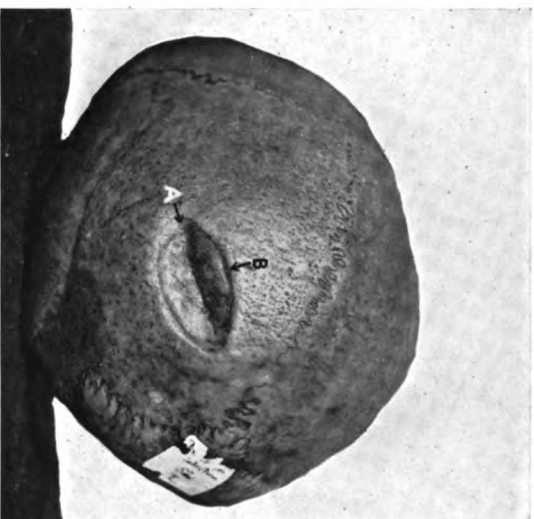


Fig. 13.

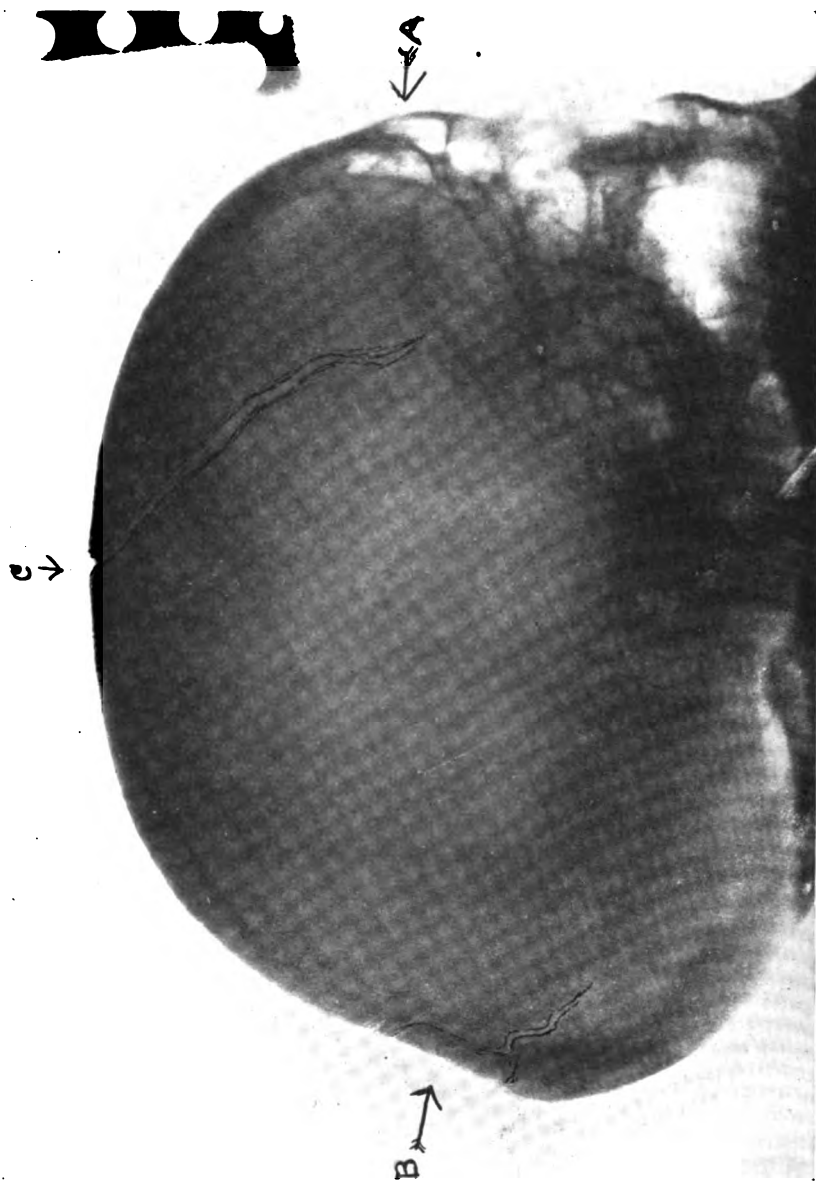


The so-called ping-pong fracture of the skull so often found in children of the lower East-side in New York City (copied from an article in the *N. Y. Med. Jour.*).

The appearance on the external surface of the skull of a fracture the result of a blow from some sharp object as an axe. The external table is fractured in two places, at *A* and at *B*. This type of fracture is illustrated in Fig. 9b. See the next figure for the effect of a blow of this character on the inner table. (Cornell Collection.)

The inner surface of the skull shown in the preceding photograph. It verifies the mechanical principles of fracture production as illustrated in Fig. 9. It shows how the fracture of the inner table first appears at the apex of the convexity of the depression of the inner table. (Cornell Collection.)

FIG. 14.



This is a radiograph of the skull of Miss F. It shows the fissured fracture at C, the result of the compressing forces applied at A and at B.

is continued, the fracture of the vault follows. Such fractures are very dangerous from the fact that after the fracture does take place, the force continues and the brain substance itself is severely lacerated and disorganized.

The above are the mechanics of the usual forms of direct violence exerted upon the skull.

There are many indirect methods of fracture production. For instance, a fall upon the buttocks may cause a ring fracture around the foramen magnum. The force is transmitted through the vertebral column, which is stationary and the skull is driven down past the foramen magnum and a fracture occurs at this point.

A fall on the back part of the head may be transmitted to or over the entire skull and cause a separation of the bones at the suture lines, such as at the lambdoid suture, the coronal or the sagittal.

A fracture, of a type which it is almost unbelievable could occur, is one described of the orbital fossa, with no evidence of any foreign body driven through, and with the eye-ball unruptured. In such a patient, the history was given that the child had been struck upon the eye. As a consequence the blow was transmitted to the orbital plate. The bone at this point was fractured and was driven into the frontal lobe of the brain and was subsequently removed at the time of operation for an abscess in this location. The eye itself, beyond some slight ecchymosis, showed no evidence of injury. (Fischer.)

Fractures of the base of the skull also occur in persons who are struck forcibly upon the lower jaw (Pugilist's Fracture). Such fractures in two instances that I know of have been the cause of death from basilar hemorrhage and cerebral compression. (Personal communication from Doctor Saling.)

*Magnitude of the Force.*—The degree or extent of the fracture of the skull, as was explained in the preceding paragraphs, depends not only upon the direction of the force, but also upon the magnitude of the force.

*Degree of Resistance.*—It is easy to understand that a blow of great severity produces much more damage than does a lighter blow, also that a blow upon the head, when the head is immobilized and held rigid, will produce more damage than when the head is freely movable.

*Elasticity of the Skull.*—The extent of the injury to the skull from a blow depends also in considerable measure upon the degree of the elasticity of the skull. In children the elasticity is much greater

than in adults, so that a fracture of the skull is not as common in infants and growing children as it is in adults. As the child grows older the proportion of lime salts in bone increases and the bone becomes more brittle. It has also been observed that fractures of the skull occur more frequently in certain areas of the skull.

*Area of Skull Involved—Diffusion of the Force.*—If we view a skull by transmitted light, we see a reason for the apparent selective localization of fractures—to certain areas of the skull. In a skull, illuminated by placing a lighted electric bulb inside the skull, we notice there are areas where the light is transmitted better than it is in other areas. These areas of better transmission of light indicate that the skull is thinner than it is in those areas where the light is not transmitted so well (Fig. 15).

On viewing such a skull we may note that on lateral view there are four areas where the light transmission is not so good. If we examine the skull more closely we notice that the dark areas are in contact with strong buttresses of bone and are themselves thicker than are the adjacent light areas. These dark areas form the supporting arches of the skull. The most anterior light area is the first portion of the frontal bone. Back of it is the thickened bone, which by transmitted light is darker than the adjacent bone. This thickened bone extends directly upward from the supra-orbital arch with an extension from the lateral margin of the orbit. The latter (the orbit) is the first great buttress of the skull. If a blow is received over the frontal bone, it is diffused downward on either side over the arch of bone corresponding to the dark area of bone and is arrested at the supra-orbital and lateral orbital margins, where it is diffused among the bones of the face.

Back of this anterior arch of bone is a little area of thinner bone, and posterior to it again we notice an area of dark bone, which corresponds to the great wing of the sphenoid. This projection of the great wing of the sphenoid also joins the first transverse arches that the force of blow received upon the first transverse arch is also diffused into the sphenoid bone. In addition to the above, we notice that directly back of the transverse arch is a lighter area of bone, which includes in its centre the suture between the frontal and the parietal bones. Back of it is the second transverse arch, which is very thick and has a projection directly backward over the occiput, practically to the foramen magnum. Posterior to it, we also notice

**FIG. 15.**



**This is a view of the skull by transmitted light. The light areas indicate that in these regions the bone is of lesser density.**





extending down over the squamous portion of the temporal bone and over the posterior surface of the parietal, the third light area. Back of and then below it, corresponding inferiorly to the mastoid portion of the temporal and laterally to the posterior margin of the parietal bone, where it comes into contact with the occipital, is the third dark area, corresponding to the third transverse arch.

If we examine the skull further, we see that there is a dark area of bone extending from the superior nasal prominence directly back over the centre of the head to the margin of the foramen magnum. This forms a very good longitudinal arch, being buttressed anteriorly on the frontal bone, and the plate of the cribriform bone and posteriorly being supported by the margin of the foramen magnum, which in turn rests upon the spine; thus, any blow received upon the top of the head would not only cave-in the transverse arches, but would also have to drive inward the longitudinal arch. The force of this blow would radiate in two directions, both longitudinal, that is, in an anterior-posterior direction, and laterally to either the right or the left or to both sides.

Each of the transverse arches in succession tends to limit both the anterior or the posterior propagation of the force radiating towards it. Fractures are most apt to occur at the point where the dense bone of the arch comes into contact with the lighter adjacent bone; that is, at the point where the transmitted force meets a resistance. The same thing happens if the blow is received on one side of the head. The longitudinal arch tends to inhibit its transference across the mid-line from one side to the other. These arches with their tendency to inhibit the conduction of the force may account for the peculiar fractures we sometimes see, in which with violence is applied to the head on one side; the effects of the blow are apparently in a direction oblique to the force of violence.

With the above evidence, we can easily see and understand how, when compression is applied upon the vortex, either by means of a sudden blow or a slow, compressing force it causes a lateral fracture in the light area of the bone. Likewise, when an antero-posterior compression of the skull occurs it is easy to understand why a fracture usually takes place in the squamous portion of the temporal, or along the coronal suture.

A sudden blow acts differently, in that the force has a tendency to be transmitted directly along the arches, which lies in the same general direction, and is interrupted only when it meets an arch running transversely to it (Fig. 14).

It is easy to understand why, in certain conditions, a blow upon the skull does not produce a fracture at a distance from the point at which the blow was received. This is apt to occur if the blow is received over a small area of the skull, and if it is not of sudden impact (application).

A fracture may either be of the outer or of the inner table.

#### FRACTURE OF THE INNER TABLE ALONE

A fracture of the inner table alone is explained from the fact that as the bone is depressed at the point of contact, the inner table is bent in more than is the outer table, until a point is reached where the cohesion of the molecules of the inner table is overcome and fracture occurs.

This fracture is fissured and, as a rule, is linear in direction (see Fig. 9 A). In such fractures the inner table springs in and the two margins separate as the bone is depressed (see Fig. 13). Some of the dura may be forced into the space between the fragments of the inner table and be torn. On springing back into place, the fragments may tuck the dura in between them, such cases have been seen and recorded. In fact, I have the picture of one such skull in which the dura extends up through a fissured fracture and appears on the external surface of the skull, underneath the muscular aponeurosis and above the periosteum (Doctor Schwartz's collection).

#### FRACTURE OF OUTER TABLE

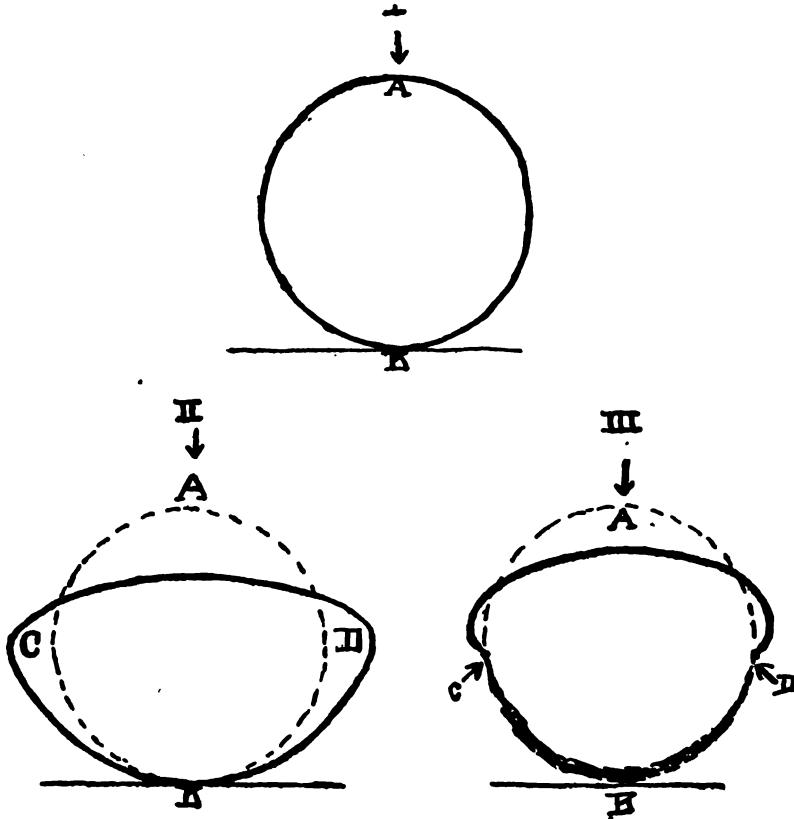
A form of violence in which the outer table may alone be fractured is that resulting from a spent bullet, which after transversing the cranial cavity, strikes against the inner table of the skull, but has not sufficient force to perforate, but enough force to bulge it outward and to fracture.

#### COMPRESSION FRACTURE

I would like to direct attention further to the point that I have indirectly suggested, when speaking of the transmission of force along the arches of the skull, but have not emphasized; namely, that if we

have a circle and exert pressure upon it at one point, and the opposite side of the circle is held firm, and shall label the point of pressure, A- and the point 180 degrees, directly on the opposing side of the circle -B; we shall find that if a force is now applied to -AC the result would be that -A would approach -B- and cause a bulging at points 90 degrees distant from A and B, or at C and D, so that the

FIG. 16.



The above drawings illustrate in a diagrammatic manner the effect which a blow received upon the top of the head will have upon the supporting arches. For a discussion of the mechanical principles involved see the text.

tendency would be for the circle to be elongated at points C and D, and to assume a more or less elliptical form; the ends of the ellipse would be at C and D (Fig. 16).

If the skull were perfectly uniform in its construction and consistency and a sufficient force were received at -A- on the vortex, the

fracture would occur at the points C and D, or 90 degrees from the place where the force had been applied. But as the skull is not uniform, either in consistency or in structure, fracture does not occur usually at such a point, but generally at some point in the arch supporting the stress. This point is usually the place where the bones of greater strength and consistency meet bones of lesser strength and consistency.

If a blow is received on the vortex of the skull, sufficient to depress the skull, so that bulging occurs on both sides and the force is continued long enough and is of sufficient intensity, fracture frequently occurs immediately above the temporal bone or in the portion of the parietal immediately above the temporal. As a rule, the fracture runs in the direction of the force.

## PHYSIOLOGICO-PATHOLOGICAL DISCUSSION IN A SYMPOSIUM ON THE ACUTE ABDOMEN \*

By ROBT. A. KEILTY, M.D.

From the Department of Laboratories and Research Medicine, The Geisinger Memorial Hospital, Danville, Pa.

---

A SYMPOSIUM on the acute abdomen must always be of interest to most of the different groups of medical workers. To the surgeon and internist, it is of importance because it is one of their daily problems, each individual case presenting its different points for diagnosis.

To the laboratory worker, it is of interest from the viewpoint of diagnosis and especially the physiologico-pathological correlation of its details. To this later part may I direct your attention during this consideration. The function of the pathologist is not only to recognize and describe the actual gross morbid changes occurring in a given case but to attempt the reasonings of those changes, the nature of their development, their stimuli, their course and their final outcome.

The acute abdomen has several objective signs and subjective symptoms which are common to all types of change. It is by a careful study of these signs and by weighing their relative predominance and importance that differential diagnosis is completed. For example, pain, tenderness, rigidity, fever, nausea, vomiting and leucocytosis might be a symptom complex of one of many different conditions. By careful study of the character and position of the pain, the degree of tenderness and rigidity, the presence or absence and degree of leucocytosis, an exact or nearly exact diagnosis is possible.

*Pain.*—Pain is a sensory response to a stimulus. This stimulus may be a factor applied at a given point in concentration, as in the development of a suppurating focus with bacterial and tissue toxins; it may be the application of physiological solutions either normal or in excess, after loss of a protective mechanism, as the gastric ulcer

---

\* Read before the 17th Annual Meeting of the 18th Censorial District of the State Medical Society of Pa., Sept. 10, 1920, Danville, Pa.

or cancer in the presence of hyperacidity, or it may be the diffusion of a toxæmic material acting directly or indirectly on sensory terminal or involuntary muscle fibres with spasm. As a basic rule the degree of pain is equal to the degree of the stimulus minus the psychico-neurasthenic factor. The localization of the pain may be either at the point of the stimulus or it may be referred to some distant point. This depends entirely upon the nerve supply and in the abdomen upon the spinal arc or arcs involved. The pain may be diffused or focal in character. This depends in part upon the spinal arc and, to a greater extent, upon the character of the reaction to the stimulus. For example, a localized, clean-cut ulcer of the stomach is painful at one particular spot just over or at the ulcer, while suppuration of the gall-bladder due to surrounding inflammatory reaction is diffusely painful over a considerable area.

The pain of a gall-stone passing along the bile ducts is severe at the point of passage and is also referred to a distant point under the shoulder-blade. This is a reflex sensation by way of the spinal arc implicated with an overlapping of the nerve supply, which is intimate at the spinal ganglia and at higher levels in the cord. Pain which is early severe may become later a complete anæsthesia. This is due to the destruction of delicate sensory terminals and even degeneration of nerve fibres as a result of the toxæmic and necrotic action of the stimulus.

Pain may be spasmodic in character. This may be due to the intermittent application of the stimulus with sensory reaction or to contractions or relaxations of involuntary muscle fibres. Later in the development, these primary contractions may become permanent dilatations, as in intestinal ileus, and then be absolutely devoid of pain.

Painful sensations may arise at one point and because of the complication of arrangements of organs be confused as to its source. This is especially true of abdominal conditions. There is a type of abdominal sensation spoken of as abdominal angina analogous to angina pectoris. In this condition the abdominal vessels, the seat of fibrotic changes and due to vascular spasms, produce painful sensations which may be distributed to most any quarter of the belly cavity. These fibroses are extremely common, and there is no doubt that they are responsible for many cases where at operation a nor-

mal appendix is found, or a gastric ulcer fails to appear and yet the patient demonstrated a majority of symptoms for the condition so diagnosed.

Before leaving the subject of pain there is a type of pain met with by the general practitioner described some years ago by McAlister and that is a "damn bad pain." This is not pathological so far as I know and is apparently quite real. It is found by practitioners mostly after long trips to the country and usually in the small hours of the night.

*Tenderness.*—Tenderness follows the general course of pain and is governed by exactly the same factors which rule the destinies of pain. It, too, is a sensory response to irritant stimuli and differs only in being more diffuse and less severe than the pain. In the case of referred pain it is not associated at the distant point.

In the abdomen there are two distinct types of tenderness—that which is persistent and becomes worse upon increasing pressure and that which is relieved by a steady, even, increasing pressure. The first is encountered when inflammatory reaction has taken place in the abdominal structure involved, and the second is due to the presence of intestinal spasm augmented by gaseous accumulations.

The rigidity of the abdominal muscles in the acute abdomen is a progression of sensory messages to the motor neurons and response equal as in the case of pain to the degree of stimulus. The motor fibres being stimulated, muscle function, which is contraction, occurs and rigidity results. The distribution of the rigidity follows in a like way the spinal-arc pathways involved. There are many abdominal pains without rigidity, that is, sensory stimuli are set up which do not terminate in motor reaction, but, where the latter are present, the original factor must be assumed to be an actual organic change. Rigidity of the abdominal muscles signifies inflammation beneath. This statement is unqualified except for certain very specific instances such as tetanus and tonic spasms due to organic nervous troubles. Wherever the peritoneum is irritated, even by the presence of a sterile hemorrhage, sensory stimuli are set up which result in abdominal rigidity. This is one of the most important objective signs, if not the most important, of peritoneal involvement in the acute abdomen.

*Fever.*—Fever in a general way is a non-specific reaction on

the part of the heat mechanism both central and peripheral. In its relation to the acute abdomen, its response is due to inflammatory reacting stimuli and of these, bacteria are the most prominent. This is, in all probability, a bacterial protein response in the blood.

Much has been written upon fever, and to date a considerable conflict of opinion remains. The nature of a fever is an inordinate production of heat over radiation and loss. The cause in abdominal lesions must be due to either the direct stimuli of bacterial bodies and their products upon the heat centres in the brain or to the protein materials produced by the body's cellular reactions. The question, is fever a beneficial body reaction in the combat of infection or is it a favoring factor for bacterial growth, is of the greatest importance. The two views are held by equally good men. The majority of therapeutics is directed at the combat of fever, and by these men, it must be considered as an element to be avoided. The older thoughts, that any fever produced degenerative changes in parenchymatous organs, must be modified. The influence of bacterial toxins is, by far, much more potent than the higher temperature *per se*. I cannot help but feel that moderate increases of temperature, up to 102°F., are beneficial, and serve to hasten immune body reaction, and should therefore be at least allowed to continue rather than be checked. Abnormally high temperatures, 104°F. or higher, are without doubt detrimental *per se* and should be combatted. It is perfectly true that in spite of treatment an infection which is being controlled by the immune factors' development in the body speedily shows a drop in temperature coincident with improvement. Let this be an appeal: In an individual case study the temperature curve carefully lest procedures directed against it do more harm than good.

The presence of fever, with the flushed face, sordes on teeth, dry lips, coated tongue and other concomitant signs, must be looked upon as an objective sign in the presence of abdominal symptoms, indicative of abdominal inflammation. Its absence, however, does not rule out such a lesion but is a factor in favor of a non-inflammatory stimulus. Subnormal temperature and its relation to shock in the advanced abdominal conditions, especially late peritonitis, strangulated hernia, intestinal obstruction and pancreatitis should be clearly recognized. In these instances the reaction is very complex, is more generalized in its nature and is always a sign of grave danger.



*Pulse.*—The pulse, an indicator of the central and peripheral blood-vascular mechanism in the acute abdominal conditions, is commensurate with the general reactions in the early stages of the disease. This response is in the intrinsic cardiovascular mechanism; the stimulus is a bacterial or chemical protein within the blood, acting directly upon the blood-vessel walls and cardia.

This is manifested by an increase in the pulse-rate which being due to the same stimulus factor as the temperature reaction is comparable to it. So, for every degree of temperature, there is approximately an increase of ten cardiac beats. As the disease progresses the irritant action of the foreign protein is augmented by its degenerate action. Myocardial degeneration advancing, alters the quality and character of the pulse, changing the entire bodily attitude. The rapid pulse of late abdominal disease is a very grave sign and its prognostic value of great importance.

*Nausea and Vomiting.*—Nausea and vomiting accompany most of the acute abdominal conditions, and, like the other signs and symptoms, vary in type and degree according to the position of the lesion and the character of the reaction. Vomiting is a very complex mechanism and is produced by a series of movements of the respiratory, abdominal and gastric muscles. The vagus also plays an important role in initiating the stimulus, the whole controlled by the so-called vomiting centre not far from the respiratory centre in the medulla. This centre is frequently stimulated directly by the action of toxins. During the act of vomiting the glottis is closed with the diaphragm held in a low position. The pyloric orifice is tightly contracted, the cardia relaxed and in some cases antiperistaltic waves occur in the stomach. At the time of the first deep inspiration some of the stomach contents are expelled into the oesophagus. Finally the abdominal muscles contract, intra-abdominal pressure is markedly increased with expulsion of the gastric contents into the mouth.

The explosive vomiting of retention in intestinal obstruction is the overflow of a paralyzed mechanism and *per se* indicates the gravity of the situation.

*Leucocytosis.*—The figures generally accepted for the normal white blood count, 5000 to 6000 are given for the normal adult. A pathological leucocytosis is an increase above this point but should

not bear much weight unless the 10,000 mark is passed. In the acute abdomen a leucocyte count of 15,000 to 18,000 should be looked for. In those bloods presenting a higher number, some complicating condition such as pneumonia may be present. In cases where pus formation has ensued the special increase will be found in the polymorphonuclear neutrophils. The leucocyte count may be taken as a great aid in the diagnosis of acute abdominal conditions, and in a large majority of cases will be indicative of the amount of immune reaction occurring, while in others that bacterial infection is present. In a few cases leucocytosis may be present from some other factor and on the other hand a failure on the part of the leucocyte due to the poor reacting quality of the patient may occasionally occur in the presence of a severe bacterial invasion. Much reliance should be placed upon the leucocyte count but it should not be considered the absolutely deciding factor.

*Discussion.*—Summed up in a few words the explanations of the generalized acute abdominal signs may be stated as follows. Some factor, and it is frequently mechanical, alters the normal abdominal physiology allowing the introduction of a bacterial stimulus. The body's reaction is exactly comparable to the degree and severity of the stimulus operative. The position and clinical reaction correspond to the group unit involved and are recognized by the alterations of its physiology called symptoms and signs.

It is needless to say that the early recognition of these alterations and their correct interpretation make the proper diagnosis. After the reaction has passed the stage of recognition of early altered abdominal physiology and has become a generalized degenerative and even necrotic reaction, the correct diagnosis is not only more difficult but often impossible. The damages of the later stages are irreparable and recovery improbable.

It is not my intention to give the differential pathology of diseases of the acute abdomen but the generalized reaction of an acute case might be of importance. These underlying factors may be applied to a disease of any organ, the signs and symptoms depending upon the altered physiology of that organ.

An appendix which has been resting in the right ileac fossa, without ever having given evidence of its being, suddenly comes to light with a pain over its region. What occurs? Its blood supply

has been interfered with, and an active congestion results. This eventually becomes a stasis with an exudation of fluid, cells and precipitation of fibrin into the lumen, wall and upon the peritoneum about the appendix. Thus, a true inflammation is established and when seen such an appendix is red, swollen, painful and warm, the old rubor, tumor, dolor and calor. By this time bacteria, ever present within its lumen, invade the process and add their reacting factors, an increase in transudative fluid, exudative cells and fibrin. This takes place in a few hours, the patient complains of pain in the right fossa due to the inflammation *per se* with an associated fever, nausea and even vomiting, the latter are entirely reflex. Upon examination tenderness localized to the site of the lesion is elicited, often the right leg is flexed upon the abdomen relieving somewhat the strain and thus the pain and tenderness. The rigidity localized to the right side is the crowning feature of the picture. Upon examination of the blood a leucocytosis of about 10,000 to 11,000 is found. At this point, or as soon before as possible, the most favorable time for operative interference presents itself. Following the principles of a disease curve the acme of an early acute exudative appendicitis is now reached.

Progression from now on may be towards complete recovery or, as occurs in many cases, the reactive element of some phase of the bacterial invasion predominates and grave complications ensue. If the congestion of the appendiceal artery becomes a thrombo-arteritis with complete occlusion, infarction in the wall of the appendix in the presence of *B. coli* produces gangrene of the wall with rupture, and a spill of the contents into the sensitive peritoneal cavity. The same result takes place where kinking of the distal portion of the organ is severe or where a foreign body, often an appendolith or a small gall-stone, acts as a pressure factor within the lumen. Frequently the invasion of the appendix by *B. coli*, which produces a necrotic reaction in tissue, will be sufficient to break down a large area of the wall and with the formation of gas accomplish a rupture. On the other hand, if organisms belonging to the staphylococcic and streptococcic groups invade, suppuration and abscess predominate. While this is not complicated by actual gangrene and rupture, the suppurative process involves the entire wall, fills up the lumen and plasters the peritoneal surface. The latter factor results in a local-

ized peritonitis, the so-called walled-off abscess, but, who can tell that this will remain walled off and will not become a generalized peritonitis either by rupture, extension or necessary operative technique? The end result in advanced cases must be a progression with eventual death or a final healing by absorption of all acute reactive elements and their replacement by fibrous or scar tissue (in the belly adhesions). This course may be decidedly influenced by early operative procedure.

The milder reactions of a catarrhal nature are the cases which never go on to gangrene, suppuration or rupture but subside almost as they begin. Who can tell in the beginning which case is catarrhal and which case is showing the early manifestations of later suppurations?

I have given very briefly the course of an acute case of appendicitis. Each reaction in the belly, whether it be a suppurating gall-bladder, volvulus, intussusception, acute entero-colitis, acute exacerbation or chronic intestinal parasitism, has in a general way acute inflammatory reactions which vary only in the intensity of the infection, the site of the infection, and the alterations of the physiology involved. A careful study of a given case along these lines constitutes the pathologico-physiological correlation of the case, and upon this basis only can a clear cut diagnosis be made, and of necessity the proper therapeutics instituted.

**THE SURGICAL KIDNEY**  
**A SYSTEMATIC METHOD FOR DETERMINING THE DIAGNOSIS**  
**AND MODUS OPERANDI**

(Continued from previous paper)

By **G. S. FOSTER, M.D.**

Surgeon to Notre Dame Hospital, Manchester, N. H.

and **S. MILLER, M.D.**

Urologist

THE discussion of the Surgical Kidney will be continued under the classification previously outlined.

**6 Traumatic.**

- (a) Rupture.
- (b) Ecchymosis.
- (c) Hemorrhagic.
  - (1) Controllable.
  - (2) Non-controllable by ordinary methods.
- (d) Markedly displaced.
  - (1) With hæmaturia.
  - (2) Without hæmaturia.
  - (3) With physical signs and symptoms (subjective and objective).
  - (4) Without physical signs and symptoms (subjective and objective).

Traumatism affecting one or both kidneys may vary very markedly in the degree of injury. It can be safely assumed that many times, injuries to the lower and middle back, either from a fall or directed blow, will affect the kidney more or less. The fact that in the ordinary individual a fair amount of fat surrounds the kidney has much to do with its not being directly involved by injury more often than it is.

On the other hand, the free blood-supply which the kidney has would tend to promote a certain amount of hyperæmia in rather slight injuries. The protective covering of the kidney and the lower border of the bony thorax serve their protective purpose very well. Nature has been most kind in our anatomical construction.

If the kidney is so injured that free hemorrhage occurs and there is communication with the general peritoneal cavity, it produces similar symptoms to those of internal hemorrhage. If the hemorrhage is free, we get marked pallor, rapid pulse, moist skin, prostration to the point of collapse. Very often the pain is acute with considerable swelling of the lumbar region. This pain may or may not radiate along the ureter into the testes or thigh of the affected side. Possibly the most characteristic sign of injury to the kidney is hæmaturia. We find the urine intimately mixed with blood and worm-like clots as casts of the ureter itself. No doubt the passage of these clots through the ureter causes the colicky pains so often seen. This hæmaturia is often most marked immediately following the injury and gradually disappears unless the injury be very extensive.

Now and then operative interference has shown extensive lacerations of the kidney without any hæmaturia. These cases are rare but have occurred, and generally the ureter is found blocked by a large blood-clot.

#### A. RUPTURE

Rupture of the kidney may or may not lead immediately to severe signs and symptoms. Such injuries as: The kick of a horse, fall from a height, gun-shot wounds or direct blows by falling objects may severely rupture this organ.

The extent of rupture of the kidney also has much bearing upon the immediate and subsequent condition. Then again we see kidneys where the capsule is intact with a rupture of the cellular kidney structure for half an inch or more. In this instance the hemorrhage is variable and controls the amount of capsule distention and hæmaturia. If the kidney cellular structure is ruptured near the lower pole the liability of much hemorrhage is less than when it occurs at the middle portion or upper pole.

When rupture occurs and the kidney is at the same time displaced, the condition resulting is apt to be more severe than when this organ retains its position. When the kidney capsule is ruptured at the same time we are very apt to get free bleeding into the general peritoneal cavity, and unless immediate assistance is given, dire results are very apt to follow.

*Diagnosis.*—The history of the case has much bearing in this instance. Immediate disability, rapid rise in pulse-rate, pallor, air

hunger, moist skin, and later a marked prostration indicate severe internal hemorrhage.

The presence of hæmaturia is very suggestive. Pain in the lower back and radiating down the corresponding ureter into the testis and thigh suggest the passage of clots down the ureter. There may be a swelling, more or less marked, at the lower back, anteriorly in the upper quadrant of the abdomen or in the flank of the affected side. A little later, œdema is very apt to be present in the skin over the lower back.

Marked tenderness over the lower back, upper quadrant of the affected side or in the flank is very often present. At times hæmaturia will form and can be definitely outlined by palpation and percussion. At times, when stones have been present in the affected kidney, their dislodgement may cause hæmaturia.

The urine is smoky and of high specific gravity. The presence of strings of blood is almost pathognomonic of hemorrhage from the kidney substance. These clots are casts of the ureter where the blood clots. If this hæmaturia persists in the absence of more grave general signs and symptoms the rupture is evidently slight and within the kidney capsule.

At times the ureter is entirely severed and in this instance or where a large clot occludes the ureter, hæmaturia may be wanting.

If the symptoms gradually abate and the presence of blood in the urine gradually lessens and later entirely disappears, the rupture is probably intra-capsular and not extensive.

The presence of a mass in the flank or upper quadrant of the abdomen indicates hæmaturia. This may or may not gradually increase in size depending upon whether or not the hemorrhage persists.

Cystoscopy will often render much assistance in clearing up the diagnosis providing the ureter remains patent. Catheterization of the ureter is often of great aid, while X-ray will often outline the limitations of involvements and assist in determination of the best method of procedure.

*Differential Diagnosis.*—The history is very important and will generally serve to rule out other conditions which lead to hæmaturia, the leading sign of ruptured kidney, cystoscopy, X-ray, examination of the blood and catheterization of the ureter will generally serve to rule out other conditions.

*Procedure.*—The majority of ruptured kidneys are surgically operative, although the degree of rupture and the resulting degree of hemorrhage govern this point. In the average case, expectant treatment should be instituted and due observations made. If the hemorrhage is free and producing exsanguination, early operative interference for the purpose of checking the hemorrhage is necessary. On the other hand, if the oozing is slight in degree and the patient responds well under rest and expectant treatment, it is best to wait, for possibly the hemorrhage will check itself and healing take place without operative interference. About one third of the cases will care for themselves in this manner.

The writer recalls two cases, one from a fall from a height and the other the result of a kick from a horse. In each instance, the rupture was severe and the blood let free so shelled out the kidney that it was distended beyond recognition. Operative procedure was instituted some weeks following each of the accidents. The patients were in extremis and nothing but firm packing of the kidney shell could be accomplished. The arterial and venous oozing was beyond control, and both patients died following repeated transfusions and all surgical interference possible.

The hard thing to decide is whether it is best to operate. Two things should govern this decision; first, the amount of hemorrhage, and second, the general condition of the patient. After the incision is made it is not always possible to do a nephrectomy as should be done in all severe cases where the kidney tissue is destroyed from pressure of the accumulated blood. Sometimes, the kidney has become many times its normal size and to do nephrectomy is formidable and unwarranted. To merely pack, tie and endeavor to control the hemorrhage in this way is all that the patient will stand.

Then again pressure necrosed tissue will cut with the suture and tying is practically impossible in many cases. The best we can do is to pack the shell and transfuse the patient and use therapeutic hemastatic methods. At times enough kidney tissue is left to secrete some urine and thus interfere with direct hemostasis to a marked degree.

The decision for doing nephrectomy in those cases that will stand such a procedure should be governed entirely by the amount of kidney tissue left. Thus it sums up that in severe cases when the patient is in extremis the best we can do is surgical amelioration and



therapeutical hemostosis. In the milder cases where extreme shelling out and distention are not so marked, nephrectomy is the method. When at least sixty per cent. of the kidney tissue is left the area should be cleaned up and suture attempted with superimposed packing. This will serve well in the milder cases. The degree of injury to the kidney governs its removal, and the amount of blood lost and general condition of the patient serve to decide whether any surgical procedure is justified. Thirty per cent. of kidney ruptures will recover without any operative interference. However, we should never wait too long, and each case is a personal equation in itself in what proper surgical judgment serves well.

#### B. ECCHIMOSIS

An ecchymotic kidney is rather hard to diagnose. It generally follows slighter injuries and gives no worrying symptoms. Following the injury we get slight pain referred to the affected kidney. This is generally a dull ache in the lower back. There is not the sharp pain occasioned by the passing of clots down the ureter. Some œdema may occur over the kidney area as a result of injury. At first, also there may be considerable blood present in the urine. This will gradually decrease in amount and soon disappear. The individual kidney function will many times be subnormal for a week or two. There is an absence of any marked degree of shock. The patient responds well to rest and administration of urinary antiseptics.

Cystoscopy will determine the kidney injured and the presence of blood following a history of direct blow or strain accompanied by the dull ache in the kidney region will generally suffice to make a proper diagnosis.

Injuries sufficient to cause ecchimosiis of the kidney tissue or capsule, or both, as is the general form, will very often be bilateral. This is quite easily determined by the cystoscopic examination and individual ureteral catheterization. A lowered bilateral kidney function immediately follows, and the injury is partially pathognomonic.

Ecchymotic kidneys recover without surgical interference. No operative procedure is warranted except in cases where the X-ray demonstrates the concurrent presence of stones. In these later cases surgical interference should be inaugurated following complete recovery from the primary injury, unless earlier procedure is indicated by continued irritation from the stones which is exaggerated by the

injury itself. In such cases early operative interference gives best results even in the face of traumatized tissue.

Ordinarily this class of kidneys should be left untouched. The patients put at rest and proper therapeutic measures instituted. The majority will get perfectly well under this form of treatment.

#### C. HEMORRHAGIC

Here we are dealing with a type of kidney that comes on insidiously, the result of continued high blood-pressure, direct result of a blow or as a sequence to other diseases like typhoid fever or other continued debilitating conditions. Pernicious anæmia, secondary anæmia, purpura, hæmophilia or the like may bring forth this type of kidney. Specific conditions as lues may cause a hemorrhage kidney.

The direct signs pointing toward a diagnosis of this type of kidney give a urine which persistantly shows the presence of blood in larger or smaller amounts. Occasionally there is present some œdema over the lower back and a dull ache which is centralized about the kidney area. The patients come to us complaining of this ache and a feeling of ill being. They are not up to the standard, and they may rehearse recent occurrence of some slight injury or strain.

Röntgenograph may show the presence of stones in the kidneys or kidney. Cystoscopy will reveal the presence of blood from the ureter of the affected side or not infrequently from both ureters following an injury or strain.

Individual kidney function will show a lowered degree of kidney activity on the affected side. Pressure over the affected kidney causes a dull pain which persists for some time following release of the pressure.

The constant presence of blood in the urine over a considerable period without or with a history of injury, lowered individual kidney function, persistent backache, and a dull pain upon pressure will warrant one in thinking of hemorrhagic kidney.

However, it is not always easy to diagnose this type of spongy, soggy kidney due to hemorrhagic oozing resulting in general limited pressure, necrosis and maceration, casts, kidney epithelium, and leucocytes may or may not be present in the urine.

*Differential Diagnosis.*—With a history of injury preceding, the continued presence of blood in the urine is suggestive. We cannot rule out the concurrent presence of stones without the Röntgenograph. Tuberculous infection cannot be ruled out without the most

severe scrutiny. The absence of repeated attempts to show tubercle bacillus in the urine is not always conclusive of the absence of this disease but will give more weight to the hemorrhagic theory. This is especially true when a history of direct blow is given. Many times it is quite impossible to rule out a malignancy.

*Procedure.*—The larger majority of these cases are non-surgical but, however, now and then a hemorrhagic kidney is surgical. Especially is this true when the presence of blood in the urine is very persistent over a long period. We have the two varieties of this pathological entity to consider.

(1) Controllable.

(2) Non-controllable by ordinary methods.

The first type is always non-surgical and should be turned over to the internist.

The second type more often warrants surgical interference. This is always true when stones are present, when tuberculosis cannot be definitely ruled out and whenever there is thought of malignancy. The larger percentage of non-controllable, hemorrhagic kidneys warrant operative interference. The true hemorrhagic kidney showing persistent presence of blood in the urine is most difficult to diagnose, under the most favorable circumstances. History of a direct blow at onset aids much yet we may find concurrent tuberculosis, stones or malignancy not always barring sclerosis.

These cases should be operated upon, and the diagnosis thus cleared up. Whether or not nephrectomy should be done depends upon the macroscopical and microscopical findings. In any case of doubt, nephrectomy is the method of procedure, always of course, first determining the status of the sister kidney. Operative procedure serves best to clear up the case, the extent depending upon the findings at time of operation.

#### D. MARKEDLY DISPLACED KIDNEY

As a result of a blow, fall or other injury, the kidney may be markedly displaced. The degree of displacement will depend upon several factors; first, the character of the blow or fall; second, the stature of the patient; third, whether or not the kidney was in good position and normally retained previous to the accident; and fourth, the amount of retroperitoneal fat present to support the kidney affected thus limiting the degree of displacement.

We take the stand that all displaced kidneys are in the altered

physiological stage, but we cannot class them as all pathological. Some kidneys which are freely movable within a wide range following an accident may give no symptoms at all.

On the other hand a kidney only slightly displaced at time of injury may yield marked symptoms and signs even to the point of so flexing the ureter as to entirely obstruct it. This may produce severe pain, renal colic or even simulate Detl's crisis.

The diagnosis of these cases is most important. Not only do we mean the making of a diagnosis of a displaced kidney following an accident, as this is not often difficult. The difficult part in making the diagnosis is the synchronous declaration of the amount of pathology produced by such a displacement. This truly is an important thing to decide as the *modus operandi* in each instance depends entirely upon this one thing. Thus we must be most careful in our survey of these cases. Not all of these cases are to be operated upon by any means, and in many instances operation, if done at all, should be delayed until all vague signs cannot be placed at the door of other conditions co-existing. In many instances, when other pathology cannot be found to account for these vague symptoms, operation to anchor the kidney will give relief. However such a result will not come forth in every instance although the percentage of reliefs is sufficient to warrant this procedure after due consideration and following proper observation.

(1) With hæmaturia.

The one thing we look for first following an accident where the kidney is displaced is the presence of blood in the urine. This will not follow in every instance of displaced kidney however. In three cases where we have noted on examination, previous to any injury, that the kidney of one side or the other was displaced and so recorded and later these cases came under our observation following an accident no hæmaturia resulted.

In each of these cases the signs and symptoms centered about the kidney area. There was a dull ache in the lower back, some frequency of micturition for a week following the accident, superficial evidence of a blow over the kidney area, as shown by ecchymosis, and more or less sharp pain radiating to the thigh of the affected side, this pain being relieved by recumbent posture and rest, but aggravated when assuming the upright position or walking about.

Whether or not this has any bearing upon the cases in general as they appear, we cannot state from our experience but we have acquired the feeling that if hæmaturia was not present following an accident when displaced kidney was found at the time of examination, that the displacement anti-dated the accident and thus the blow or fall merely centered the signs and symptoms about this already altered anatomical kidney.

In other words, we believe that a kidney already displaced to a marked degree can pass through the effects of an accident and not bleed or ooze merely because it can readily be pushed aside from the direct influence of the blow and is therefore released from any extensive injury producing hæmaturia. However, this may be, observations by others along this line would assist materially in establishing the stability or instability of the fact.

When we have a markedly displaced kidney following an accident and hæmaturia is present we divide our cases into three groups, namely: Those with clear, fresh blood in the urine so plentiful that it masks all other things that might be seen and interferes with proper cystoscopy and catheterization of ureters while the patient shows general signs of rapid loss of blood; those cases where the hæmaturia is very marked presenting proper sediment examination; cystoscopy or ureteral catheterization yet the patient shows no signs of shock or collapse; those where the hæmaturia is slight and does not increase and the patient is in general good condition. We let this classification govern our judgment as to recommendations.

In the first class we always operate at once and immediately follow the operation by a transfusion. This to be repeated if necessary. Just what is done at the time of operation depends entirely upon what is found. If the kidney is very large and shelled out by the hemorrhage and the kidney has been severely shattered we do a nephrectomy if possible. If the patient's condition will not warrant this step we merely pack the kidney shell and surrounding areas very snugly, clamp or tie any apparent points of bleeding just before packing. The patient is then given some systematic hemostatic and transfused. We then wait and in a few days after the patient has sufficiently recovered from the shock we remove the kidney. This can be done through the same incision as a rule but now and then has to be delivered per anterior abdominal wall, but extra-peritoneally if possible. Occasionally, the transperitoneal route has

to be used. Whatever the route, deliver this class of kidney as soon as possible and thus control the hemorrhage.

In the second class of cases we put the patient at complete rest and use systemic hemostatics and await results. If the pain becomes more severe and the strength of the patient is taxed, after proper observation we do a nephrectomy and if necessary follow the operation by transfusion. If the patient progresses well, and the hæmaturia lessens, yet the tumefaction gradually increases we do a nephrectomy. In all of these cases where nephrectomy is ultimately done we have felt that we might have aided the patient if it had been done earlier. On the other hand if the hæmaturia gradually disappears and the tumefaction which was present, gradually recedes, we await final results. If the urine entirely clears up, and the patient gradually returns to normal, we consider any surgical interference unnecessary. However, we keep these patients under observation for one year, doing a very frequent urinalysis and an occasional cystoscopy and catheterization to determine the individual kidney function with regular precision.

In the third class we put the patient to bed and following a urinalysis daily, cystoscopy and ureteral catheterization, and determination of individual kidney function prolong the observation until all signs and symptoms have cleared up. However, if hæmaturia persists and the sediment shows no organismal growth or smear presentment, we take under advisement co-existing tuberculous kidney which was aggravated by the accident. If the question of tuberculosis cannot be safely ruled out, we think best to operate, after proper observation, and, if the kidney tissue is involved, to do nephrectomy. This seems to us the safest route. Of course the great majority of this class of cases get well without operative interference.

SUMMARY OF TRAUMATICALLY, MARKEDLY DISPLACED KIDNEY, WITH  
HÆMATURIA

*Class 1.*—Operate at once and do nephrectomy at first sitting if possible. If not, do this after recovery shock. Always support with systemic hemostasis and transfusion.

*Class 2.*—Operate in the face of persistent hemorrhage and marked dilatation and shelling out of kidney tissue. Nephrectomy is the operation to elect and when facts are conclusively determined the earlier the surgical interference the better. If signs and symptoms abate, operation is not warranted unless the displacement itself

is producing aggravation. In this event we do not operate until complete recovery from the shock of the accident, possibly some weeks or even months later.

*Class 3.*—The greater majority of these cases fully recover without any operative interference. Ruling out a co-existing tuberculous condition, operative interference is not warranted unless such be recommended some weeks or months later merely to fix the kidney with the hope of relieving some vague symptoms still persisting. Not all vague symptoms and signs will abate even with this procedure, yet some, although but few, will entirely clear up.

(2) Without hæmaturia.

Not infrequently we see patients with a markedly displaced kidney following an accident and no presence of hæmaturia. At no time during the course of convalescence does blood appear in the urine. The kidney is markedly displaced, easily felt and freely moved from place to place. At times we have seen these kidneys become more or less firmly fixed following the accident. We have thought this sign due to adhesions. Even with the history of a blow or fall and at time of examination finding a markedly displaced kidney but no evidence of hæmaturia, there was no proof that the displacement did not anti-date the injury. This would be a difficult thing to prove unless we had a previous physical record of the case.

Of course in each instance, providing the patient is informed of the displacement, the most natural thing is to award the accident the cause of such displacement. If we leave no proof that the kidney was displaced previous to the accident then we cannot deny the statement.

There are two classes of cases under this heading, namely, (a) with physical signs and symptoms (subjective and objective); (b) without physical signs and symptoms (subjective and objective).

(a) *With Physical Signs and Symptoms.*—This type of kidney is the one which is most apt to confuse us in making a proper diagnosis in so far as the accident is concerned. The patient passes through the accident and subsequently develops a confused area over the lower back or in the buttock region. A dull ache in the lower back soon shows itself and possibly some pain radiating down to one thigh or the other. This pain is not very severe in the average case. However now and then we have met a patient who related the story of rather acute, piercing pain radiating to the thigh, lasting a short time and then gradually or suddenly disappearing with change of

posture and leaving a dull ache in the lower back. Sometimes a frontal headache will be complained of, dull in type and rather persistent for a week or sometimes longer. Frequency of micturition will come on at this time and last from three to ten days, this disappear. At times the frequency of micturition and rather acute radiating pain persist for a long time.

Physical examination reveals a freely movable kidney which is many times hypersensitive to pressure of rather slight degree. This kidney is generally quite freely movable, and if watched for a time may later become more or less limited in excursion by manipulation, and radiograph, cystoscopic examination and catheterization of ureters may or may not show a slight curve, kink or constriction in the ureter. Possibly, the recognition of the latter condition, namely a slight constriction in the ureter may account entirely for the pain. The character of pain in these cases is generally dull and rather persistent. If any alteration in individual kidney functions exists that can be determined.

If the signs and symptoms persist after attempt at relief through diet, alteration in posture, rest or mechanical support, operative interference is warranted. It is just this class of cases disregarding the injury, that get a good deal of relief from proper fixation, and the majority are fully amenable to operative interference.

(b) *Without Physical Signs and Symptoms* (subjective and objective).—Here we see a patient who has passed through an accident without complaining subsequently of the kidney area. There may be evidence of injury elsewhere even to the point of fracture, dislocation and the like yet the kidney is not referred to in any way, and the urine is found negative in every respect. On physical examination, however, the kidney is found displaced and very freely movable. The mobility of the kidney may decrease as time goes by due to the formation of adhesions. Here again, we meet the problem of whether or not the kidney was displaced previous to the accident, perhaps for years past. The probability is in favor of the latter condition.

This type of case does not warrant any surgical interference for cystoscopy, catheterization of ureters, individual kidney function test and radiograph show nothing abnormal except the displacement.



We are not justified in operating these cases except under three conditions, namely, if the patient is aware that such displacement previously existed or does now exist and demands fixation; in the case of vague signs and symptoms not accounted for by other pathology or altered physiology; the subsequent development of acute, sub-acute or chronic conditions from which relief might be expected by such a procedure.

We can only consider this last type of traumatic kidney surgical in the case of demand on the part of the patient or from later developments. However dietetic, postural and mechanical support measures should be instituted in an endeavor to replace the kidney. It so happens that not infrequently, that type of kidney, if displaced, at time of accident, will be quite amiable to some of these later named measures, than supporting the belief that it occurred at time of the accident. Such measures should be instituted rather early before adhesions form to tie the kidney down in displaced position. In case such measures are not successful in securing the desired results, we must feel that the probability is that the kidney was displaced previous to the accident. Then we are more truly warranted in considering operative interference. Especially is this true in the face of such a desire on the part of the patient.

#### TUMORS OF THE KIDNEY

Under this head we have numerous growths which are found to involve the kidney. However, tumors of the kidney are relatively infrequent and the majority are malignant. The Massachusetts General Hospital reports 74 cases of tumors of the kidney covering a review of the histories for a period of 40 years. Up to 1912 only 83 cases had been seen at the Mayo Clinic. A review of the hospital reports of St. Bartholomew's, London; Bellevue, New York and Mayo Clinic; Lakeside, Cleveland, shows that renal tumors constituted but 14 per cent. of all tumors of the urogenital tract. At St. Lukes' Hospital, New York, for the past 12 years, there were 325 cases of kidney disease (excluding nephritis), of which 24 were tumors. This shows the relative infrequency of kidney tumors, compared to tumors elsewhere in the body.

##### (7) *Non-malignant Growths.*

(a) *Involving the Kidney Parenchyma.*—Under this heading we

find but few growths of the kidneys *per se*. Among those tumefactions found in the kidney, not suppurative, hemorrhagic or obstructive are cysts. In this class we find simple serous cysts, dydatid cysts, dermoid cysts, and polycystic degeneration and adenomata. We may also more rarely see lipoma, fibroma or angioma, Now and then rhabdomyoma may be found. We may also find mixed tumors of the above classes which many times exist with non-malignant growths.

(b) *Extra Capsular*.—These growths are even more rare than those involving the parenchyma of the renal structure. However, we do occasionally see lipoma, fibromata, angiomata and small cystic accumulations that are found entirely outside of the kidney capsule but adherent to it. Now and then a hyperplasia of the suprarenal body is seen and glandular enlargements of a more or less harmless and insignificant form. The diagnosis is made as a rule, upon three findings, the presence of a tumefaction in the region of the kidney, complaint of pain over the lower back or hypochondriac regions, and hæmaturia. The tumefaction may or may not reach extensive dimensions and may or may not be freely movable. It is more often found very large in infants or people over 60 years of age. Pain is complained of as existing in the lower back. Now and then so-called renal colic is present, which is generally occasioned by obstruction from a particle of tissue or blood-clot. Hæmaturia may or may not be present. In the non-malignant types excluding the angiomata and possibly the rhabdomyomata, it is rare.

The patient may complain of one, two or even all of these symptoms. No infrequently in these growths very few symptoms are brought forward except under the complaint of not feeling well.

Cystoscopy, catheterization of ureters, individual kidney function test and urinalysis are many times very helpful in making a differential diagnosis tentatively at least between the malignant and benign growths. Radiograph may help somewhat in single instances, but as a rule, it is not of much assistance. Injection of the pelvis and radiograph of the catheter *in situ* is occasionally of some material assistance.

These kidneys are all surgical and warrant operative interference. Nephrectomy should be done when the individual kidney function is

found below 15 per cent. or 60 per cent. of the kidney structure has been destroyed. If resection of the growth is possible, and enough functioning tissue can be left conservatism should be made.

When the growths are extra capsular, resection is the method to be elected. If at any time suppuration has complicated the growth as may be found in the cystic forms, glandular enlargements, etc., drainage should be paramount.

(8) *Malignant Growths.*

(a) *Involving the Kidney Parenchyma.*—The majority of renal tumors are malignant. As such their existence should be recognized very early and relief instituted at once if we are to expect the best results. Under this head, we find hypernephroma which constitutes 80 per cent. of all solid tumors of the kidney. It has been said that 90 per cent. of all tumors appearing after puberty in the kidney are hypernephroma. Recent years have shown by the close observers that these growths are malignant beyond any doubt. Possibly in the very beginning this status cannot be definitely determined by the microscope but later there is no question of its malignancy. Metastases will quite often occur in the bones, lungs and liver.

Carcinoma of the kidney is relatively infrequent but probably, as stated by Swan, constitutes at least 2 per cent. of all malignant renal growths. This growth may extend to the ureter and bladder. Very early we are apt to get a softening and breaking down of the capsule. Carcinomata do not become very large as they are usually fatal in the early stages.

Sarcomata of the kidney, which occur more often in children, on the other hand are softer in consistency and generally nodular. They are not infrequent and sometimes grow to be enormous in size before causing death.

Adenomata are occasionally found in the kidney tissue, and are of the malignant type, although the malignant, as frequent as the non-malignant, fibular, alveolar and papillary forms are found, of which the latter are most frequent.

Occasionally we see angiomatous degeneration of any one of these malignant growths and the cavernosum formation is also seen, while mixed cell-growths, highly malignant and early in metastases are also found.

In making the diagnosis, the same methods are instituted as in the non-malignant forms. Hæmaturia is a very prominent sign and when persistent, or regularly intermittant, seems quite suggestive in conjunction with tumefaction, persistent dull pain and loss of weight and strength. Individual kidney function determination is most important. Here, as in the non-malignant forms, radiograph in conjunction with catheterization and pelvis injection may help to a certain extent but fails many times in its conclusiveness. Early diagnosis of malignancy of the kidney is the important thing.

(b) *Extra Capsular*.—Adenomata and metastases, although rare, are now and then found. Any malignancy near the kidney soon involves the renal structure itself. They are very apt to pass recognition until later when the kidney structure has become involved. Malignancy of the suprarenal gland is found occasionally but not often recognized as such.

Early operative interference is the method, the earlier the better and the more radical the operation, the better the result, complete through dissection and separation of the kidney and the full length of the ureter.

Nephrectomy *in toto* including the ureter as far as the bladder blocking often all possible avenues of contamination and checking all bleeding points at once.

The final results over long periods of observations reveal cases of non-remissions although metastases to the bladder and sister organ are liable or even to more distant parts. Direct extension to the genito-urinary tract in general is very common.

Very early diagnosis and operation give the best results. The trouble lies in the fact that most patients wait in the development of the disease.

#### (9) *Accumulation of Secretion.*

The kidneys are now and then affected by the accumulation of secretion within itself or beneath the capsule. This may be due to hyperactivity on the part of the cells with resulting walling off of a sectional area of the kidney, causing single or multiple cysts, or it may occur from some obstruction with one or more tubules of the kidney. Accumulation of this secretion may also affect the ureters from either constriction secondary to malformation or scar tissue, or from mechanical obstruction as blocking from a calculus within its

lumen or pressure from a growth without a twisting. While these various forms of secretory accumulation are not very frequent, yet there findings are seen and results noted occasionally.

(a) *Accumulation in Ureter.* Accumulation of urine within the ureter may come from several causes. A calculus may become lodged within the lumen of the ureter and obstruct it. In this instance we occasionally see a markedly distended ureter which will resemble a sausage in confirmation. If the obstruction continues for any length of time, the ureter may become greatly distended. Such an obstruction generally comes on rather suddenly and is secondary to the stone or stones in the kidney. One or more stones have passed from the kidney pelvis down the ureter and its size has prevented continuous passage. This generally causes very severe pain of the Dietl's crisis type. This pain radiates to the corresponding thigh and is of the lancinating type. It is often remittant as nature endeavors to dislodge the stone. At times, various amounts of blood or no blood may be found in the urine. Radiograph will show the stone in the majority of instances. Catheterization of the ureter and radiograph of the catheter *in situ* will clearly show the location of the stone. Individual kidney function will assist, possibly nil on the affected kidney. Cystoscopy will reveal the amount of urine passing down the affected kidney. The radiograph may show shadow outlining the tumefaction from distention of the ureter. At times large amounts of urine gather in the ureter.

Now and then cases of a moderately distended ureter are seen caused by obstruction due to pressure from some tumefaction in the abdominal cavity. Pressure from a large ovarian cyst, lipoma, fibroids or infringement by some malignant growth which presses upon, or surrounds and constricts the ureter. Often these forms of obstructions do not cause great ballooning of the ureter but they may be seen. They are very apt to occur rather intermittantly and between times large amounts of urine are passed, and frequency of micturition may be present and occasionally a small amount of blood is seen in the urine. This form of trouble shows itself by intermittantly irregular pain, rather dull in character and radiating down to the thigh and scrotum. Cystoscopy and catheterization of the ureter will help very materially in clearing up the cases. They are generally insidious in onset and the history covers a long period. Many

times the intra-abdominal tumefaction can be clearly demonstrated by abdominal or, in the case of women, by-manual examination. Rectal, digital examination often times assists in clearing up the case.

Recently much has been written regarding congenital strictures occurring in the ureters of which trauma may be a cause. It is not often that they will so materially occlude the ureters as to cause ballooning, yet this has been found. Generally when this does occur it is secondary to trauma or a malignancy.

A kink in the ureter or a twist may cause obstruction temporarily, persisting until relieved by manual interference. A very marked ballooning of the ureter may result which suddenly disappears and is followed by the passing of a large amount of urine. This may repeat itself, fairly frequently or may occur but once and disappear. In these cases if the obstruction is only partial and the constriction, due to scar tissue or a congenital malformation, does not balloon the ureter, but there is present a persistent dull pain radiating to the groin and few remissions of pain more or less sharp in character. Blood may or may not be present in larger or small amounts. Cystoscopy, catheterization of ureters, radiography, and determination of the exact point of constriction by a picture of the catheter *in situ* is of great assistance.

(b) *Minlocular Cyst Formation.* Simple singular retention cysts are not frequent but now and then we run across them. They are possibly more often found when performing some other operation in the kidney region, during exploratory laparotomy or post-mortem. These singular retention cysts are caused by some chronic inflammatory condition in the kidney as in chronic interstitial nephritis or in some other kidney lesion where the tubules or lymph channels become occluded or pressed upon, or neoplasms or calculi in the kidney may cause this condition. Many times the tendency is to become multiple rather than remain single.

We seldom get any referable signs or symptoms unless the cyst becomes quite large and encroaches upon the capsule. In this instance, we do get a dull pain from the distention of the capsule. They are a matter of curiosity rather than a surgical entity as such.

Solitary cysts have been seen in kidneys which are otherwise normal. Von Brackel has found 21 cases gathered from literature and Tuffier has collected 38 cases. Ordinarily only one, generally

the left kidney, is affected, and the cyst is most often found near the lower pole of the kidney. The majority of these cases are in females. Some believe these cysts to be congenital, and others place the cause at the door of tight lacing or the like. Infection of these cysts may follow. Sieber reported 200 cases; 10 per cent. of which ultimately became infected.

Not often do these cysts become large although they have been found very large and palpable through the anterior abdominal wall. When they are large enough to cause pressure they give rise to much pain, at times of a colicky nature. If they become infected, pus may be found in the urine if they communicate with the tubules. Unless very large they do not give rise to symptoms and remain for years without doing harm.

(c) *Multilocular Cyst Formation*.—Polycystic degeneration is the type of cystic kidney most often seen, and unfortunately this disease is apt to be bilateral. These kidneys become very large often three or four times the size of normal kidneys. The surface of the kidney is irregular and nodular. The cause of the condition is not clear. Virchow considered the cause as inflammatory within the kidney tissue resulting in very many tubules becoming occluded and forming retention cavities. Some argue they are congenital. Possibly the congenital arrest of development has much to do with their formation.

In conjunction with these cysts we are very apt to get a fibrosis occurring between the cystic walls. This type of kidney is found most often in infants. Some writers, as Garceau and Senator, consider the disease more common to middle life. Sieber collected a series of cases numbering 173 in all. He found that the greatest number occurred between the ages of fifty and sixty years. These cases have occurred in a series of individuals of the same family which supports the congenital theory.

Binney states that about 98 per cent. of the cases are bilateral. When unilateral he states that the left kidney is the one most frequently involved.

The prognosis in infants is grave, generally proving fatal. If the patient is an adult, the outlook is much more favorable. As the parenchyma of the kidney is not encroached upon to the extent of other kidney growths the excretory function can remain relatively

sufficient to continue life. When the disease is unilateral the patient often goes through life unhampered, death coming from some other cause.

Pain, dull in character and rather persistent in the renal area, is a symptom. In some cases however we do get renal colic of the radiating type, at times bilateral and quite severe. This is, however, more often remittant at long intervals. These conditions may at times simulate stone in the kidney but the radiograph will be of material assistance in ruling this out. The urine will show at rather long intervals remissions of hæmaturia which persists for a certain period and then disappears to later reappear for a certain period. Such a phenomenon will cover several years in many cases. As the disease advances renal changes show in the urine, and subsequently a typical chronic nephritis develops. Anæmia and a bronzed skin are very apt to appear later in life. The cardiovascular changes of renal diseases follows. We may get the marked loss of weight simulating malignancy, loss of strength, headaches, dysponia, etc. If continued uræmia comes on, and then death.

When the kidneys become very large the tumefaction is easily felt through the anterior abdominal wall. Cystoscopy may show blood coming from the ureter of the affected side and radiography may reveal an enlarged shadow area.

However the diagnosis is never easy. With the ever present possibility of its being bilateral any unilateral trouble should go through a very rigid differential diagnosis before the polycystic kidney is decided upon. The presence of a large tumor in the kidney region, intermittant hæmaturia, pain when bilateral and intermittant will assist. However it is seldom that a true diagnosis will be made preoperatively except when bilateral tumefaction is present in the kidney region. This makes such a diagnosis more likely. Secondary infection of some of these cysts may occur and pus may or may not be present in the urine.

(d) *Obstruction at the Pelvis.*—This condition in the kidney is not wholly unknown. There are various causes for the obstruction but the result is practically the same. The extent to which the kidney becomes enlarged or dilated depends upon the completeness of the secretion accumulating behind the obstruction. Obstruction at the pelvis of the kidney or at the very beginning of the ureter bringing



about an accumulation of urine in the pelvis and kidney, calices and tubules is termed hydronephrosis. This condition may be congenital or acquired, caused by abnormal conditions without as well as within the kidney or ureter. It may also be continuous or permanent or it may be intermittent or remittant.

A stone ledged tightly at the beginning of the ureter is the most frequent cause of this condition. Some foreign growth within the pelvis or upper ureter or compressing the upper ureter or pelvis from without may bring on the same condition. Some obstruction may be present at almost any point along the course of the ureter as some congenital or acquired structural defect constricting the lumen, an arrested calculus, a large organized blood-clot or a tumefaction within or without causes a complete obstruction. At times a very large prostate or some growth in the bladder may cause such a complete obstruction.

Binney states that the majority of hydronephrosis cases are produced by factors which exist at birth. Among these he states that the remains of valves which normally exist in foetal life just at or below the uretero-pelvic junction may bring about obstruction. Malformation of the ureter, movable or horseshoe kidney, a congenital stricture of the ureter or an anomalous blood vessel which passes over the upper part of the ureteral tube and diminishes its lumen may bring on this condition. Excessive effusion in the abdominal cavity or even ligation during an intra-abdominal operation may cause hydronephrosis. This condition is generally unilateral. We find pressure, atrophy, marked anæmia and later permanent atrophy of the parenchyma resulting. When this is of standing, the kidney becomes a shell filled with the retained material. We find a compensatory hypertrophy of the sister organ.

Now and then a bilateral hydronephrosis will develop from back pressure caused by some growth in the bladder of constriction secondary to inflammatory conditions in the bladder and here the bladder and both kidneys will share in the hypertrophy.

*Diagnosis.*—This is not difficult as a rule. A large, palpable tumor in the region of the kidney shows fluctuation and with a more or less lengthy history is very suggestive of hydronephrosis. Occasionally a large ovarian cyst may simulate this condition. Cystoscopy, catheterization of the ureters, determination of individual

functional test, pyelography following collargol injection, radiograph, urinalysis and the use of an exploratory needle will generally make the diagnosis of this condition clear,

*Prognosis.*—If the condition be unilateral and no complications are present the prognosis is generally favorable. Bilateral existence of this condition makes the outcome fatal in the majority of instances. Aunisia, uræma, pyonephrosis and rupture of the sac when they occur, make the outlook very grave.

*Treatment.*—This condition always warrants early surgical intervention. The earlier the surgery the better the result, and the majority of instances nephrectomy should be done at once. However, if the dilatation or blocking is due to a very movable kidney and 40 per cent. of the kidney tissue is intact, correction of the displacement will often times result favorably. The function of the opposite kidney should, as in all other kidney conditions, be determined pre-operatively. However, even a poor sister kidney will so hypertrophy as to do very good work following unilateral nephrectomy.

If the condition is caused by an aberrant blood-vessel, this can be ligated and divided and will give the desired results if the kidney parenchyma is 40 per cent. intact. A stricture of the ureter may be treated plastically or dilated and will often give relief although this is not always wise as it may recur.

If caused by a calculus this may be removed and if the parenchyma is 40 per cent. intact. At times the stone may be so situated as to act as a valve at the ampulla. In this case we may get a sudden disappearance of the tumor accompanied by the passing of a large amount of urine and frequency of micturition. This may be remittant or intermittant and only partially or completely ameliorative for the time. Pyelectomy and removal of the stone will give the desired results in the majority of instances.

In the average case nephrectomy is the method of election. However we should always conserve where possible, and if nephrectomy, nephropexy or some like procedure will suffice this is all that is warranted. Early surgical intervention is to be accomplished in all cases.

Therefore under the general sub-heading of accumulation of secretion we make a summary as follows:

## SURGICAL

*Nephrectomy*

1 Unilocular cyst formation (if below 40 per cent. parenchymatous involvement).

2 Multiple cyst formation.

3 Hydronephrosis (when not corrective or parenchyma below 40 per cent. intact).

*Nephrotomy*

1 Unilocular cyst formation.

2 Hydronephrosis (when removal of obstruction is possible and parenchyma 40 per cent. intact).

*Nephropeasy, or Other Correction.*

1 Obstruction in ureter (either without or within).

2 Hydronephrosis.

(a) Displaced kidney.

(b) Aberrant blood-vessel.

(c) Calculus in ampulla or below (providing parenchyma is 40 per cent. or better intact).

(10) *Hemorrhage With the Kidney Capsule. Non Surgical.*

1. *Unilocular cyst formation (no symptoms existing).* There are instances of hemorrhage within the kidney substance itself or beneath the capsule and not involving the kidney structure. Binney has termed the presence of blood in the urine without any definite cause, "essential hæmaturia." It would seem proper to assume that in any instance where there is blood present in the urine some pathological or at least abnormal or unusual physiological condition must be present. In other instances hemorrhage within the kidney tissue or beneath the capsule or both can be attributed to a definitely known pathological alternation. These conditions will be taken up under two headings.

(a) *Severe and Distending (Acute).*—Under the heading of traumatic kidney we have dealt with that type of kidney which is flooded with free blood. These kidneys are always surgical and immediate. Now and then we see a kidney where there is present single or multiple varix, and the vessels become eroded, over distended and ruptured. This condition will give rise to very profuse hemorrhage at times, and the kidney tissue itself will under go pressure necrosis from the accumulation of a large amount of confined blood.

The varix, one or many, may be situated just beneath the capsule and produce a marked accumulation of blood causing a marked distention of the capsule and at times a very large kidney result. Often

under these circumstances blood will not show in the urine and the only evidence we have is the presence of a tumefaction over the kidney area if the amount of oozing is sufficiently large to greatly distend the capsule. This tumefaction is generally symmetrical although it may be irregular, rather hard and not fluctuating.

A chronic of infectious nephritis may produce a diffuse hemorrhage in the kidney tissue. At times the hemorrhage may very markedly distend the capsule and produce a rather large, regular tumefaction.

In hæmophilia we occasionally see a kidney water-logged by diffuse hemorrhage. Here sub-capsular hemorrhage will at times produce a very large kidney. The blood should always be carefully examined and coagulability time determined. If hæmophilia is known to exist in the family, this as a cause should always be seriously considered.

Secondary infections as by the colon bacillus or other organisms may produce diffuse hemorrhage into the kidney substance or sub-capsular hemorrhage.

Inflammatory or passing congestion alteration in the glomeruli may bring on a subcapsular hemorrhage by burrowing or a diffuse and marked hemorrhage into the kidney tissue. Hyaline degeneration of the blood-vessels, persistent high blood-pressure, thickened, sclerotic capsule, granular pyelitis, interstitial lesions of the pyramids, tumors, varicosities of the renal capsule and breaking down of malignant growths may produce this same diffuse and sub-capsular hemorrhage with varying degree of distention.

*Symptoms.*—Ruling out such conditions as malignancy, trauma, chronic nephritis, etc., the symptoms are not prominent except one, the presence of blood in the urine. Very little if any pain is present unless a blood-clot in the ureter causes colic.

*Diagnosis.*—Many times this is very difficult. Unless the cause is demonstrable such as malignancy, nephritis, sclerosis, persistent high blood-pressure, etc., it may be impossible to make the diagnosis. Minute hemorrhage, sub-capsular may be present for some time without being recognized. Hemorrhage into the kidney tissue or beneath the capsule is so often merely a forerunner or adjunct sign of other trouble that it should always be taken very seriously. The hemorrhage itself may prove fatal although this is rare.

*Treatment.*—All medicinal measures, rest, diet, etc., should be thoroughly tried before any surgery is suggested. However if the hemorrhage persists and shows in the urine, or if the kidney gradually increases in size, surgical intervention is warranted. Any persistent hemorrhage into the kidney tissue or sub-capsular, if not controlled by ordinary means, warrants exploration by surgery.

When the kidney is delivered, it should be very carefully examined and if the cause is located, it should be resected, ligated or otherwise treated depending upon its nature. In any case where the capsule has become greatly distended, decapsulation should be done, the clots cleaned out, and the kidney anchored. If the hemorrhage persists during the procedure, and the blood elements are normal, or have no shown any improvement under medication, nephrectomy should be done.

Malignancy, tuberculosis, sepsis, infections, or the like warrant nephrectomy as a rule. This gives the best results. Of course it goes without saying that all measures should be instituted previous to any procedure surgical in nature, to demonstrate the ability of the sister organ.

Where there is a large accumulation of blood and no demonstrable pathology is seen, it is best to be conservative. Often drainage of these conditions will give the desired results. If 40 per cent. or more of the kidney tissue is destroyed, nephrectomy should be done. In any instance, conservative treatment of the kidney, first medically and then surgically, should be followed out as warranted. In event of failure by these methods, nephrectomy is the operation of election.

(b) *Gradual and Persisting.*—Gradual and persisting hemorrhage into the kidney tissue or sub-capsular is brought on by practically the same cause as innumrated under the previous heading. It is a matter of degree rather than cause.

*Treatment.*—In any instance where sub-capsular or interstitial hemorrhage has persisted for along time without causing marked symptoms or signs including hæmaturia or tumefaction exploratory investigation is warranted. If the persistent leaking cannot be controlled by definite dealing with a pathological area, one is warranted in considering nephrectomy rather seriously. However, no surgical measures should be undertaken in any instance until medicinal treatment has failed. Nephrectomy should not be done if the bleeding

can be controlled by packing or the like. If bleeding persists and all other measures, medical and surgical exhausted, nephrectomy is warranted even in the face of undemonstrable pathology.

(11) *Phlebectasis*.

Whenever a patient complains of a continuous, dull ache in the kidney region accompanied by remittant or continuous varying degree of hæmaturia, one should not overlook the possibility of distended tortuous veins in the kidney. This condition is not often bilateral, and is rather rare. However, it should not be overlooked in making a differential diagnosis. Two varieties of this condition exist.

(a) *Beneath the Capsule (Sub-capsular)*.—These kidneys are generally found at post-mortem or an ordinary exploratory laparotomy. The distended, thin-walled veins feel like worms as one passes the hand over the affected kidney. Nearly all of these cases of course are chronic. The only form of acute phlebitis, sub-capsular in type, is infective of origin and generally accompanies some other disease, as typhoid fever, scarlatina or the like. The acute form gives a picture unlike the acute hyperæmic kidney and generally subsides without further trouble.

The chronic form, which is the one we are most interested in, results from some pathological change which has so thinned the veins that they have lost their tone. Lues might be one of these causes. A general athermatous condition can exist and pathological section of the vein wall shows a fatty infiltration. Calcareous deposits are very often found in the walls of these veins. Often with a sub-capsular phlebitis nothing shows in the urine. The chief complaint is a persistent ache in the kidney region, this having come on rather gradually. The diagnosis is seldom made except tentatively. This is an entity found during exploration or post-mortem.

*Treatment*.—As a rule sub-capsular phlebectasis is not treated as such for the reason that it is not recognized unless found by exploratory laparotomy. Only very rarely the condition might be recognized by most careful exclusion in differential diagnosis.

Rarely is surgery resorted to without making the specific diagnosis the one pronounced symptom of continuous dull pain over the kidney area will be relieved by proper support, hygienic and dietetic principles and proper amount of rest. However if a sub-

capsular phlebectasis is found during an abdominal or other exploration, it is always best to intermittently ligate the veins and section between the ligations and tie together of the raw stumps. This will, as a rule, relieve the pain. Surgical principles are instituted when the affected kidney is seen but seldom otherwise. It is a surgical entity when found. The average case is treated by supports and rest however.

(b) *Interstitial*.—This form of phlebectasis is rather rare. Post-mortem kidneys are sectioned and the greatly dilated veins found. The most frequent signs and symptoms are hæmaturia, remittant or continuous, of a varying degree and persistent ache in the kidney region.

So many other kidney conditions cause these same phenomena that it is hard to make the specific diagnosis. Then again the phlebectasis may co-exist with other kidney pathology. It may also be a sequel to typhoid fever, scarlatina and the like. If it exists as a sequel, it generally takes care of itself when the cause is removed. In other words it is an acute, infective thing and has a limited run.

Even if interstitial, phlebectasis exists, it is hard to realize the fact even when the kidney is seen. The diagnosis is more or less a guess unless the kidney is split.

*Treatment*.—Support, rest, hygienic and dietetic principles will often relieve the back ache and hæmaturia if continued long enough and if no other pathology is present. If other pathology is co-existent, then it will not be relieved to the extent of curing the trouble. The phlebectasis will be helped but the other pathology not much altered.

If the kidney is split to remove stones or for other reasons the enlarged veins may be ligated, sectioned and the stumps tied together. This will relieve the phlebectasis. Ordinarily this condition is not primarily surgical. It becomes a surgical entity only when found while exploring or surgically treating other kidney conditions.

#### CONCLUSIONS

In summing up it may be stated that the majority of kidney conditions warrant some sort of surgical intervention, although there are a few kidney conditions where surgery is of no benefit.

Generally speaking early surgery, when such intervention is war-

ranted, gives best results. The condition of the sister organ should always be first determined and every other laboratory investigation made. Every precaution should be used to protect the patient against untoward results or long continued disability.

Kidney surgery is safe surgery if properly done and the right time selected. It is also most satisfactory as a rule and gives the patient the relief sought.

*Chart Tabulation Showing Surgical Kidney Summary*

Disease	Surgical	Medical	Medical and Surgical	Occasionally Surgical	Treatment
Nephritis	No	Yes	No	Yes	Medical, occasional de-capsulation.
Interstitial Parenchymatous					
Displaced Kidney (Nephroptosis)	Yes	No	No	No	Pexy; none if congenital.
Nephrolithiasis	Yes	No	No	No	Removal of stones.
Tuberculosis	Yes	No	No	No	Nephrec-tomy.
Septic Infection	Yes	No	Occasionally	No	Nephrectomy and drain-age; nephrec-tomy.
Traumatic (a)					
Rupture	Yes	No	No	No	Nephrec-tomy or re-pair.
(b)					
Ecchimosiis	No	Yes	Occasionally if infected	Yes if infected	Nephrec-tomy; drainage.
(c)					
Hemorrhagic					
(1) Controllable	No	Yes	No	No	Medical.
(2) Non-controllable	Yes	No	Yes	No	Nephrec-tomy or nephrotomy.
(d)					
Markedly Displaced					
(1) With Hæmaturia	Yes	No	Yes	No	Pexy.
(2) Without Hæmaturia	Not Always	Sometimes	Best	Yes	Pexy.



Disease	Surgical	Medical	Medical and Surgical	Occasionally Surgical	Treatment
Non-malignant Growths	Yes	No	No	No	Removal of growth; occasional nephrectomy.
Malignant Growths	Yes	No	No	No	Nephrectomy with dissection.
Accumulation of Secretion					
(a)					
Obstruction in Ureter	Yes	No	No	No	Removal of obstruction.
(b)					
Unilocular Cysts	No	No	Yes	Yes	Nephrectomy or dissection of cysts.
(c)					
Multilocular Cysts	Yes	No	No	No	Nephrectomy.
(d)					
Obstruction at Pelvis	Yes	No	No	No	Removal of obstruction
Hemorrhage					
(a)					
Severe and Acutely Distending	Yes	No	No	No	Nephrectomy or nephrotomy and drainage.
Gradual and Persisting	Not Always	Yes	Occasionally	Yes	Nephrotomy and drainage; nephrectomy.
Phlebectasis					
(a)					
Subcapsular	Yes	No	Yes	Yes	Ligation; section apposition of stumps.
(b)					
Interstitial	Not Always	Yes	Yes	Yes	Ligation; section apposition of stumps.

## BIBLIOGRAPHY

- BINNIE: "Treatise on Regional Surgery," vol. ii, 448.  
BINNIE: "Treatise on Regional Surgery," vol. ii, 398.  
*Analytic Cyclopaedia Sajo*, vol. vi, 213.  
BINNIE: "Treatise on Regional Surgery," vol. ii, 399.  
BINNIE: "Treatise on Regional Surgery," vol. ii, 408, 413, 432.

# INDEX TO VOLUME III

## (THIRTY-FIRST SERIES)

### A

- Abdomen, acute symptoms in, 273  
     fever, 274  
     leucocytosis, 277  
     nausea and vomiting, 277  
     pain, 278  
     pulse rate, 277  
     tenderness, 274  
 Abscess of prevesical space and umbilicus, 111  
 Acetabulum, fracture of, 223  
 Actinomycosis, 1  
     broncho-, 10  
     cachexia of, 61  
     differential diagnosis, 4, 15  
     lymph-nodes in, 77  
     of appendix, 59  
     of breast, 19  
         clinical aspects of, 29  
         pathology of, 19  
     of cæcum, 59  
     of lachrymal ducts, 83  
     of liver, 50  
         gastric and intestinal form of, 56  
         hepatic form of, 55  
         pyæmic form of, 57  
         symptoms of, 55  
     of lungs, 1  
     or nervous system, 90  
     of skin, 73  
     of tongue, 84  
     pleuro-, 14  
     pneumo-, 10  
     symposium on, 1  
     treated with potassium iodide, 107  
 Amputations, 200  
 Ankylosis of joints, 196  
 Appendix, actinomycosis of, 59  
     differential diagnosis, 70  
 Aribaud, George, 50

### B

- Behan, R. J., 259  
 Bérard, D. G., 107  
 Bettman, Ralph B., 126  
 Bonnet, Denis, 84  
 Breast, actinomycosis of, 19  
     clinical aspects of, 29  
     pathology of, 19  
 Bronchi, actinomycosis of, 10  
 Bronchiectasis, 17

### C

- Cæcum, actinomycosis of, 59  
     differential diagnosis, 70  
 Cancer of lung, 17  
 Children, disturbances of hearing in, 175  
 Clement, Charles, 242  
 Coulter, John S., 195  
 Cyst of kidney, 291, 299, 303  
 Cysts of urachus, 111, 119

### D

- Davis, Geo. G., 226  
 Deafness in children, 175  
 Dislocations, 196

### E

- Ecchymosis of kidney, 285  
 Ellis, John D., 229  
 Employees, examination of, 230  
 Epidemiology in industries, 232  
 Epilepsy, 255  
     tonsils and, 255  
 Eyeball, steel in, detection of, 242

### F

- Femur, internal dislocation of, 223  
     sarcoma of, 220  
 Food dreads, 161  
     value of, in neuroses and psychoneuroses, 156  
 Foster, G. S., 281  
 Fracture of acetabulum, 223  
     of inner table of skull, 270  
     of outer table of skull, 270  
 Fractures, 197  
     of radius, 250  
     of skull, 259  
         compression, 266, 270  
         mechanism of, 259  
     of vertebrae, 204  
         laminectomy for, 204  
 Freeman, Rowland G., 189

### G

- Gangrene, pulmonary, 16  
 Gastric ulcer, heart block simulating, 138  
 Glanders, pulmonary, 4

## H

- Hæmaturia, 288, 290  
 Hæmophilia, 304  
 Hammerschlag, Victor, 175  
 Hearing, disturbances of, in children, 175  
 Heart acute dilatation of, in children, 193  
     block simulating gastric ulcer, 138  
 Hemorrhagic kidney, 286  
 Hendricks, Wm. A., 204, 214  
 Hepatic actinomycosis, 50  
 Hernia, inguinal, Meckel's diverticulum in-  
     carcerated in, 126  
 Herniotomy, acute Hodgkin's disease fol-  
     lowing, 217  
 Hinglais, Manuel, 59  
 Hodgkin's disease, 214  
     acute, after herniotomy, 217  
 Hydatid disease of lung, 17  
 Hydrocephalus, radical cure of, 255  
 Hydronephrosis, 300

## I

- Industrial surgery, 195  
     physiotherapy in, 195  
     surgical clinics, 195  
 Industries, medical and surgical services  
     for, 229  
 Infantile scurvy, 179

## J

- Job, Emile, 90  
 Joints, ankylosis of, 196  
 Jopson, John H., 250

## K

- Kelty, Robt. A., 278  
 Kidney, cyst of, 298, 299, 303  
     displaced, 287  
     with hæmaturia, 290  
     ecchymosis of, 285  
     hemorrhage within capsule of, 303  
     hemorrhagic, 286  
     rupture of, 282  
     surgical, 281  
         classification of, 281  
         summary of, 308  
     tumor of, 293

## L

- Lachrymal ducts, actinomycosis of, 83  
 Lamectomy, 204  
 Liver, actinomycosis of, 50  
     gastric and intestinal form of, 56  
     hepatic form of, 55  
     pyæmic form of, 57  
     symptoms of, 55  
 Lumbar vertebræ, fracture of, 204

- Lung, actinomycosis of, 10  
     cancer of, 17  
     hydatid disease of, 17  
 Lymph-nodes in actinomycosis, 77

## M

- Magnuson, Paul B., 195  
 Malingerling, 200  
 Meckel's diverticulum, 126  
     incarcerated in hernia, 126  
 Milleff, S., 29  
 Military tuberculosis, 192  
 Miller, S., 281  
 Monestié, François, 73  
 Muscle injuries, 199

## N

- Naussac, Joseph, 1  
 Nerve injuries, peripheral, 199  
 Nervous system, actinomycosis, 90  
 Neuroses, 156  
     value of food in, 156

## P

- Pædiatrics, 175  
 Peritonitis, tuberculous, 123  
 Pfeiffer, Damon, 111  
 Phlebotaxis, 306  
 Physiotherapy in industrial surgery, 195  
     cost of, 200  
     equipment, 201  
     supervision of, 200  
     in treatment of ankylosis of joints, 196  
         of dislocations, 196  
         of muscle injuries, 199  
         of nerve injuries, 194  
         of sprains, 196  
         of tendon injuries, 199  
 Pleura, actinomycosis of, 14  
 Pneumoconiosis, 209  
 Pneumonia in child, 190  
     without fever, 191  
     without leucocytosis, 191  
 Poiteau, M., 19  
 Potassium iodide for actinomycosis, 107  
 Prevesical space, abscess of, 111  
 Pulmonary actinomycosis, 1  
     gangrene, 16  
     glanders, 4  
     syphilis, 4, 16  
 Psychoneuroses, value of food in, 156

## R

- Radius, fractures of lower end of, 250  
     reduction of, 250  
 Ravdin, Elizabeth Glenn, 138  
     I. S., 138  
 Renal veins, thrombosis of, 132  
 Reynolds, Cecil Edward, 255

Rhinoscleroma and actinomycosis, 81  
 Robert, George P., 83  
 Rupture of kidney, 282

**S**

Sarcoma of femur, 220  
     periosteal and actinomycosis, 81  
 Scurvy, infantile, 189  
 Semilunar carpal bone, dislocation of, 226  
 Skin, actinomycosis of, 73  
     differential diagnosis, 80  
 Skull, fractures of, 259  
     compression, 266, 270  
     inner table of, 270  
     mechanism of, 259  
     outer table of, 270  
 Sprains, 196  
 Steel in eyeball, detection of, 242  
 Surgical kidney, 281  
     classification of, 281  
     summary of, 308  
 Syphilis and actinomycosis, 81  
     pulmonary, 4, 16

**T**

Tendon injuries, 199  
 Thrombosis of inferior vena cava and  
     renal veins, 182  
 Thymus, enlarged, 190  
 Tongue, actinomycosis of, 84  
 Tuberculosis and actinomycosis, 80  
     acute millary, 192  
 Tuberculous peritonitis, 123  
 Tumors of kidney, 293

**U**

Ulcer gastric, simulating heart block, 138  
 Umbilicus, abscess of, 111  
 Urachus, cysts of, 111, 119

**V**

Vena cava, inferior, thrombosis of, 132

**W**

Walsh, James J., 156  
 Weber, F. Parkes, 132













med



UNIVERSITY OF MICHIGAN

3 9015 07037 2092



